

TRANSACTIONS  
OF  
The Association of  
Life Insurance Medical Directors  
of America

SIXTIETH ANNUAL MEETING

James R. Gudger, M. D.  
*Editor*

VOL. XXXV

PRESS OF  
Recording & Statistical Corporation  
New York City  
1952

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MEDICAL DIRECTORS OF AMERICA  
Printed in U. S. A.

## CONTENTS OF VOLUME XXXV

	Page
Officers, Executive Council, and Former Officers .....	iv
Opening Address by Lauritz S. Ylvisaker, M.D. .... (President's)	1
Papers:	
Cortisone, Hydrocortisone and Corticotropin: Some Facts and Speculations with Special Reference to Rheumatoid Arthritis, Philip S. Hench, M.D. ....	5
The Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease, Howard M. McCue, Jr., M.D. ....	35
Discussion .....	41
Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis, J. Gilbert Falconer, M.D. ....	43
Discussion .....	53
Current Progress in Cardiovascular Research, Francis R. Dieuaide, M.D. ....	57
The Differential Diagnosis of Chest Pain, H. M. Marvin, M.D. ....	64
The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks, Henry B. Kirkland, M.D., Charles E. Kiessling, M.D. and Annie Mary Lyle, F.S.A. ....	86
Discussion .....	131
Calcification of the Thoracic Aorta: A Mortality Study, William Bolt, M.D., and Murray F. Bell, M.D. ....	135
Discussion .....	144
The Life Insurance Examiner and the Cardiovascular System, James R. Gudger, M.D. ....	149
Relationships Between the Medical Profession and the Health Insurance Council, James Andrews, Jr. ....	162
The Public Health Situation Today: Public Health and Civil Defense, Herman E. Hilleboe, M.D. ....	171
Some Contributions of Public Health to Life Insurance, George M. Wheatley, M.D. ....	189
The Impact of Life Insurance on Public Health, Ronald F. Buchan, M.D. ....	201
The Prognosis of Benign Gastrointestinal Conditions, Franz J. Ingelfinger, M.D. ....	206
Discussion .....	231

## CONTENTS OF VOLUME XXXV—Continued

	Page
Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers, Lawrence L. McLelland, M.D., and D. Sergeant Pepper, M.D.	224
Discussion .....	231
Mortality Among Insured Overweights in Recent Years, Louis I. Dublin, Ph.D., and Herbert H. Marks .....	235
Discussion .....	263
Insurance Hazards of Overweight: Dietary Factors in the Development of Atherosclerosis, Thomas M. Durant, M.D. .....	267
Overweight as a Contributing Factor in the Development of Diabetes and Its Complications, Edward S. Dillon, M.D., and John M. Trapnell, Jr., M.D.	280
Overweight as a Contributing Factor in the Development of Hypertension, Joyce T. Sheridan, M.D., and John McC. Peck, M.D. ....	291
Discussion .....	296
Remarks by President-Elect Linford H. Lee .....	299
List of Those in Attendance .....	301
Obituary .....	304
List of Members of the Association .....	305
Honorary Members .....	324
Emeritus Members .....	324
Companies and Their Representatives .....	325
Indices: Volume XXXV .....	342
Cumulative — 1947 - 1951 .....	345

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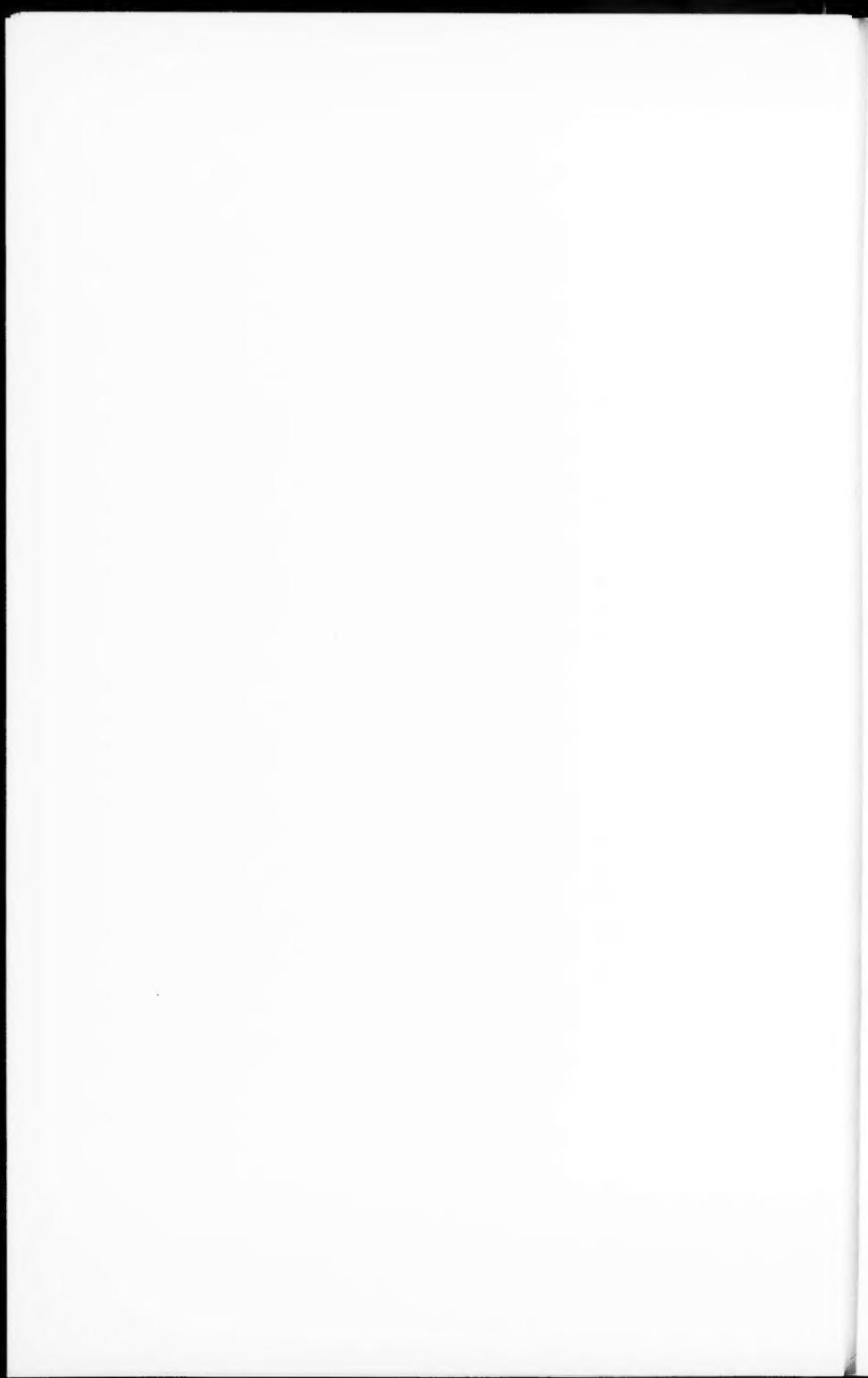
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**Transactions  
of  
The Association of  
Life Insurance Medical Directors  
of America**

**SIXTIETH ANNUAL MEETING**

The Sixtieth Annual Meeting of The Association of Life Insurance Medical Directors of America was held at the Hotel Statler in New York City on Thursday and Friday, October 11 and 12, 1951.

**PRESIDENT YLVISAKER** — It is a pleasure for me to welcome all of you to this, the sixtieth annual meeting of our Association. The completion of sixty years of activity is a significant event in the lifetime of our organization. During these sixty years, we have had many distinguished predecessors who have given the best years of their lives to build our Association to its present strength. We owe them much and we can best show our appreciation of their efforts by carrying on as they would have us.

As a result of their activities, we now have a membership of 404 life insurance medical directors representing 209 companies holding the major portion of the 250 billion dollars of life insurance in force in the United States and Canada in the hands of 83,000,000 policyholders.

It is as such a group that we are meeting here this week to formulate our plans for the future. We are conscious of the fact that we represent both life insurance and medicine and that the program we develop must be to the best interest of life insurance and in accord with medicine at its best. Life insurance and medicine are a natural team. They have similar aims. Life insurance wishes to provide everyone with economic security and the peace of mind which goes with such security. Medicine contributes to a better life by improving both the physical and mental health of the individual and his community.

Life insurance medical directors have the dual responsibility of promoting both economic security and better health, and

## SIXTIETH ANNUAL MEETING

the dual privilege of extending the benefits of both to the individual. We are all thrilled to observe the wonderful progress made by both life insurance and medicine in recent years. Inspired by these successes, we cannot fail to go ahead and solve the problems which are still before us. We can perhaps best visualize these problems by noting from the 1950 report of The Institute of Life Insurance that 280,000 individuals had to be declined for insurance last year because their health was so impaired that it was economically impossible to provide them with the benefits of life insurance. For similar reasons, 740,000 individuals had to be issued insurance at substandard rates or at an increased premium. There were considered by us over a million individuals who were impaired in health with known residuals of former disease or with indications of impending trouble.

In our examinations of presumably healthy individuals, medical directors see disease in its earliest stages. We find hypertension before this condition has made any inroads on the apparent health of the individual. We meet with diabetes before the individual is aware of its presence. We see cancer while cure is still possible. We find the earliest manifestations of arteriosclerosis while it may still be possible to delay its progress.

Although we still have as our chief responsibility the protection of our companies from unexpected mortality losses due to the impairments which we discover, we cannot be true either to the industry or the profession which we represent if these observations do not awaken in us a desire to do something about the unfavorable health conditions which we find to be still existing.

We need to make the individual and the public aware of the impairments which we detect. We should encourage medical research which will develop a full understanding of these impairments and ways and means to cure and prevent them, and we should assist in promoting every public health endeavor which will properly inform the public of the health hazards that still exist and urge everyone to do everything possible to eliminate them.

It is with these responsibilities in mind that we have prepared our program for this meeting. We have noted guest speakers who will report to us on some of the most significant developments in medicine. We hope they will be able to tell us that these developments will do much to continue to improve world health.

Our own distinguished members will discuss health impairments as they are still found to exist among those applying to us for insurance. We hope these speakers will be able to report that these impairments are decreasing in both frequency and severity, and that we are today able to insure more and more applicants because of improved health conditions among them. We are making rapid progress in that direction.

The 1950 report from The Institute of Life Insurance states that 88 per cent of those applying for insurance were accepted at the usual standard rates and that an additional 9 per cent secured insurance at extra rates. Only 3 per cent had to be declined. These figures represent considerable improvement over those of previous years. We can hope for continued improvement. We in our Medical Directors' Association can do much and should do more to encourage our insurance industry to assist in so improving health conditions that everyone can eventually enjoy the benefits of both good health and economic security.

The first guest speaker on our formal program is Dr. Philip S. Hench, Professor of Medicine, University of Minnesota Graduate School, Mayo Clinic, Rochester, Minnesota. Dr. Hench and his accomplishments are so well known to all of us that he needs no introduction.

The December, 1950, issue of *The Lafayette Alumnus* which is dedicated to him (Dr. Hench graduated from Lafayette College in 1916) gives us this interesting information — "The name of Philip S. Hench of the class of 1916 was added to the list of the world's 'immortals' in the field of medicine on December 10, 1950, on which day he received in Stockholm the Nobel Prize in the field of medicine and physiology. As

this award has come to only a few American scientists, it may be of interest to tell something of the history of the awards and the nature of the work by virtue of which they were made during the years since the first award in 1901" ....

"On a level with these discoveries is to be mentioned that of the laureates of this year, Kendall, Reichstein and Hench, by virtue of which the baffling and crippling disease of rheumatoid arthritis and certain other diseases which have troubled mankind throughout all history may be eventually controlled." ....

"The Nobel Prize was the crowning honor of the many heaped on Dr. Hench for his work for humanity. He has honorary degrees from Lafayette, Washington and Jefferson, Western Reserve University and National University of Ireland. In 1942, he received the Heberden Medal (London) for researches in rheumatic diseases.

"In 1949, he was the recipient of the Lasker Award, presented by the American Public Health Association for valuable contributions to human biology and for outstanding scientific achievement.

"In 1950, the Newspaper Guild of New York gave him the Page One Award for the discovery and development of cortisone, an achievement affecting the future welfare of mankind.

"Also in 1950, he received the Passano Foundation Award for fundamental contributions to the science and practice of medicine and the Scientific Award of the American Pharmaceutical Manufacturers Association, with Prof. George Thorn, of Harvard, in recognition of fundamental research on cortisone.

"On February 15, 1951, he will receive, with Dr. Kendall, the Award of Merit of the Masonic Foundation for Medical Research and Human Welfare."

We have with us, then, one of the most distinguished men in medicine today. He will speak to us on the very important subject of cortisone and ACTH. It is a pleasure for me to introduce to you Dr. Hench.

CORTISONE, HYDROCORTISONE AND CORTICO-  
TROPIN: SOME FACTS AND SPECULATIONS  
WITH SPECIAL REFERENCE TO  
RHEUMATOID ARTHRITIS\*

PHILIP S. HENCH, M. D.

*Division of Medicine,  
Mayo Clinic,  
Rochester, Minnesota*

*Introduction*

Three years ago today I was in London to read a paper before the Heberden Society. The title of my paper was "The Potential Reversibility of Rheumatoid Arthritis." (28) In that paper I made five points: 1. Under the influence of certain conditions, especially pregnancy and jaundice, the symptoms of rheumatoid arthritis are markedly suppressed. 2. Obviously, rheumatoid arthritis is not necessarily a relentless, progressive disease but is potentially reversible, and rapidly so. 3. During pregnancy and jaundice the body apparently makes an antirheumatic "substance X" which is probably "a biologic compound specific in nature and function" but is "neither bilirubin nor a unisexual (female) hormone." 4. Since either pregnancy or jaundice often suppresses temporarily the symptoms of several other conditions (for example, asthma, hay fever, psoriasis, Addison's disease), "substance X" is not merely antirheumatic or "disease specific" but is "group specific." 5. If "substance X" could be identified it might provide a method of control, or constitute a superior treatment, not only for rheumatoid arthritis but for several other conditions mentioned.

When that paper was written (during the summer of 1948) I had no precise idea what organ might be the source of substance X but, for reasons given elsewhere, it seemed possible that substance X might be a bisexual hormone. This reasoning had led Dr. Kendall and me to decide in January, 1941, to

\* Read at the meeting of the Association of Life Insurance Medical Directors of America, New York, New York, October 11, 1951.

try out his compound E (17-hydroxy-11-dehydrocorticosterone; substance Fa of Reichstein) against rheumatoid arthritis whenever it might become available. But we did not get to use it until September 21, 1948. Thus, it happened that just a few days before I was to leave for London my colleagues, Drs. Kendall, Slocumb, Polley, and I suddenly discovered that "compound E," which Dr. Kendall and I later renamed "cortisone" suppressed markedly and rapidly the symptoms of rheumatoid arthritis. We wondered, of course, whether cortisone might be the beneficial substance X of pregnancy and jaundice.

Seven months later we gave our preliminary reports (30, 31, 33, 41) on cortisone and ACTH: on what these hormones then appeared to be able to do and what they could not do.

#### *Summary of Our Preliminary Observations and Conclusions*

We observed the following:

When *adequate* amounts of cortisone, or ACTH now officially called "corticotropin," were given to patients with rheumatoid arthritis, symptoms markedly diminished in practically all cases. But the disease was not cured, and when use of the hormones was discontinued the symptoms returned, promptly in most cases, sometimes slowly and incompletely, but occasionally not for several months.

The various manifestations of the acute phase of rheumatic fever were usually abolished quickly, some within a very few days, others within two or three weeks.

Observations made in the spring and summer of 1949, in 1 case each of severe disseminated lupus erythematosus and psoriatic arthritis, in cases of hay fever, bronchiectasis and neurodermatitis coincidentally present in patients with rheumatoid arthritis, and in 2 cases of tuberculous arthritis appeared to indicate that cortisone or corticotropin could exert a pronounced suppressive influence on these diseases also—at least on certain of their features. (29, 32, 34) In the case of lupus erythematosus the arthritis, pericarditis, pleurisy and leukopenia disappeared temporarily during hormonal usage, but albuminuria and hypertension persisted. In the

2 cases of tuberculous arthritis, without pulmonary involvement, symptoms disappeared and sedimentation rates became normal but results of articular biopsy, guinea pig tests and cultures remained positive.

It became obvious that although the capabilities of these hormones were striking they were of a special and limited sort. In a variety of inflammatory conditions the hormones exerted a suppressive effect on symptoms, not a curative one on the disease itself. Except in certain acute, self-limiting conditions (for example, acute rheumatic fever), relapses occurred sooner or later when use of the hormones was discontinued. The hormones were not bactericidal or bacteriostatic. They appeared to act, not by removing the causes of the diseases responsive thereto, but by suppressing in large measure the reactions of the tissues to the irritating agents. Although the hormones influenced greatly the reversible part of these diseases, the pathologic physiology (the "fire," the active inflammation which causes symptoms), they exerted no influence on the irreversible part, the pathologic anatomy or residual "ashes."

It was obvious also that patients varied in their tolerance to these hormones. Although most patients tolerated fairly high doses quite satisfactorily for a few days or a few weeks, and although some patients tolerated well 100 mg. of cortisone daily for fairly long periods, from these early, rather high dosages side effects of some sort developed sooner or later in the majority of cases and were often troublesome.

*Do Cortisone and Corticotropin Act  
Physiologically or Pharmacologically?*

The effectiveness of the hormones against several diseases, rheumatoid arthritis in particular, posed for us a major question: "How do these hormones work—by correcting some type of adrenal cortical insufficiency not previously suspected of being present in these diseases? Does rheumatoid arthritis develop because there is a deficiency of endogenous cortisone or corticotropin, perhaps a deficiency in production?"

We soon had to dismiss the latter idea, at least temporarily, for the following reasons: 1. rheumatoid arthritis is rarely associated with Addison's disease; 2. currently available laboratory tests (estimation of urinary steroids; eosinopenic response of rheumatoids to an injection of exogenous corticotropin) did not reveal any definite evidence of adrenal cortical insufficiency; and 3. the doses required to suppress rheumatoid arthritis were much larger than those (10 to 15 mg. a day) which usually control Addison's disease.

For these reasons we could only conclude tentatively that if the action of cortisone was a physiologic one it was not simply a matter of replacement therapy, like the use of insulin in ordinary (pancreatic) diabetes mellitus. Such a conclusion led others (22, 72) to suggest that cortisone and corticotropin exerted against rheumatoid arthritis and other responsive diseases not a physiologic, but a pharmacologic effect. There was then no way to settle this point quickly. But one had to remember that, after all, the effective agents, cortisone and corticotropin, are not foreign substances like penicillin but are natural agents, extremely complex substances which the body actually makes and without certain amounts of which one cannot live.

We then wondered: If the effect of cortisone is not pharmacologic and not physiologic in the sense of simple replacement, could it be physiologic in a more complex sense? (16, 42) If in rheumatoid arthritis there is no obvious deficiency of production, no absolute deficiency, could there be a relative deficiency because of increased destruction, or abnormal utilization by the cells, or increased tissue need? There was then and there still is no way to answer that question either, because so much basic information is missing.

*Missing Data of Fundamental  
Importance: Unfinished Business*

Little is known about the physiology of the anterior pituitary body and of the adrenal cortex in health and disease: what hormones they produce and in what amounts, how these hormones are metabolized and how their metabolism is

regulated, how the recipient cells utilize them, and in what form and amounts and by what routes their metabolites are excreted.

*Hormonal Production: What Kinds, How Much?*—Is there only one pituitary adrenocorticotropic hormone or more than one as some physiologists suspect? How much corticotropin does the pituitary gland produce daily in a normal person under no special stress? How much of an increased production of corticotropin is required for a normal person under sudden physiologic stress or for sick people under the stress of chronic illness or under a superimposed acute stress (for example, surgical operation)? Does the pituitary body of a patient with rheumatoid arthritis, for example, produce (without artificial stimulation by exogenous corticotropin) enough endogenous corticotropin to supply him with enough cortisone and other cortical hormones? Is cortisone the only, or chief, adrenocortical hormone, or are there others, for example, corticosterone (Kendall's compound B, Reichstein's substance H) and hydrocortisone (Kendall's compound F, Reichstein's substance M) which are secreted as part of the finished product of the adrenal cortex and which may be of equal or greater clinical usefulness? How much cortisone or cortisone-like hormones do normal persons need—only 10 to 15 mg. per day? How much cortisone do sick people need daily? Regardless of how much sick people need, are their tissues getting enough?

*Metabolism.*—Once discharged into the blood stream, what proportion of these hormones is utilized by the cells (in health, in disease) and what amount, if any, is normally destroyed or inactivated? Does some organ play an intermediate role in the metabolism of cortisone, interposing its activities on the amounts of this hormone passing between the adrenal cortex and the recipient, peripheral cells such as those of joints and muscles? Does the liver function as a regulator and inactivate normally a certain amount of these adrenocortical steroids as it does estrogen? (19)

Two or three years ago there were, and even now there are, no answers or only incomplete answers to these ques-

tions. They cannot be answered until new biochemical methods have been devised, some of which may take years to develop. Therefore, it was obvious to us that in utilizing these new hormones for further studies in clinical physiology and experimental therapeutics we could not administer them in a physiologic manner. They had to be administered empirically, carefully, as intelligently as possible, with due consideration for calculated risks.

#### *Policies Governing Empiric Administration*

To govern our empiric administration of these hormones we formulated two general policies which we believe are important (32). 1. in the presence of functioning adrenal glands one must learn how to cooperate with them rather than to dominate them or to try to take over their function, and 2. until more is known about these hormones, about pituitary and adrenocortical function in rheumatoid arthritis and in the other responsive diseases, and about the control of side effects, one should not aim for complete suppression of rheumatic or other symptoms but should be content to use doses of the hormones which will give optimal (not necessarily maximal) results, the greatest relief that can be obtained without the development of significant side effects.

#### *Recent Progress and Accumulated Data*

As a result of the intensive efforts of many investigators working in many fields all over the world—the efforts of biochemists, physiologists, clinical investigators, pathologists and others—much progress has been made and much information has been obtained during recent months. I shall review this briefly.

*The Search for Substitutes for Cortisone.*—In the hope of finding a substitute for cortisone which could be made more easily, cheaply and abundantly, one which would produce comparable therapeutic effects but perhaps fewer side effects, many adrenal extracts and between 50 and 100 steroids have been tested vigorously in man and in animals by various

biologic and clinical methods. Apart from hydrocortisone (17-hydroxycorticosterone: compound F of Kendall; substance M of Reichstein) none has been found to possess significant or comparable therapeutic activity. Several of the ineffective steroids or those without antirheumatic properties differed from cortisone only in one single configuration; in fact they were cortisone except for one change.

*Antirheumatic Properties of Steroids; Specificity of Cortisone.*—As a result of these numerous clinical and biologic tests one can now define precisely the antirheumatic properties of the steroid structure: the delta 4 bond and the functional groups at positions 3, 11, 17, 20 and 21 all are essential. (42, 59) Since only cortisone and hydrocortisone fulfill, so far, such structural requirements these substances appear to be highly specific.

*Production.*—As the superiority and physiologic specificity of cortisone and hydrocortisone became more obvious, the already intensive efforts to improve the production of these hormones were greatly extended. More biochemical teams and more manufacturing chemists entered the field. As a result of prodigious effort and brilliant chemical pioneering, old methods have been greatly improved, and new methods have been discovered whereby the total synthesis of cortisone, and also hydrocortisone, can now be accomplished from a variety of starting materials—from coal tar, plants and vegetables; for example, the coal tar derivative orthotoluidine, stigmasterol from soy beans, diosgenin from Mexican yams, hecogenin from sisal waste. (10, 18, 37, 49, 60, 83) These remarkable chemical discoveries have been made very much sooner than was predicted a year or two ago. Merck & Co., Inc., the distinguished pioneering manufacturers of cortisone, have improved their methods so successfully that cortisone, priced at \$200 per gram only twenty-eight months ago (July, 1949) now (since last week) costs the patient only about \$25.00 per gram. Prices will fall further when these new methods reach the stage of mass production.

Hydrocortisone can now be made by partial synthesis from bile salts (76), by biosynthesis from Reichstein's substance S (24, 25, 55, 70) and by total synthesis. In the near future, amounts adequate for longer clinical and metabolic studies will be available.

Corticotropin is now made from the pituitary glands not only of pigs but also of cattle, horses and whales. (36)

*Preparations.*—Cortisone can now be given not only as a suspension for parenteral injection but also in tablets for oral use and in eye drops and ophthalmic ointment. A cortisone ointment is being tested against certain diseases of the skin. (64, 65) Two "long-acting" preparations of corticotropin have been developed. (21, 81, 82) The intravenous administration of corticotropin given slowly as an infusion has been recommended for special situations.

Cortisone given orally in tablets was effective in 99 of 100 rheumatoid patients studied by us. (75) Most patients required no more, or only a little more cortisone when it was given by mouth than when it was given intramuscularly. One patient, unrelieved by tablets, responded to injections.

*Physiologic and Chemical Studies.*—Important studies on the chemical structure and molecular weight of corticotropin are being made; potent peptide fractions have been prepared. (2, 43, 45, 56)

It now appears that most of the twenty-eight crystalline compounds which have been recovered from the adrenal cortex are physiologically inert precursors of a small number of active substances, among them being the compounds B, E and F of Kendall, and the amorphous fraction which has not been identified chemically. The preparations 11-dehydrocorticosterone (compound A of Kendall) and corticosterone (compound B of Kendall; substance H of Reichstein) are not antirheumatic (13, 32), but corticosterone influences mineral metabolism and probably will have a certain therapeutic usefulness.

*Relationship Between Cortisone and Hydrocortisone.*—Does the adrenal cortex discharge into the circulation both cortisone

and hydrocortisone, or only one of these compounds? Is one of them the true hormone, the ultimate secretion, and is the other merely its immediate precursor, the penultimate product? Some investigators believe that cortisone is the precursor of hydrocortisone which is the real hormone produced by the normal human adrenal gland, at least when it is stimulated by exogenous corticotropin. (13, 50, 57) Kendall (42) has expressed the belief that when the adrenal cortex is strongly stimulated (by exogenous corticotropin or by other strong stress) hydrocortisone is discharged into the blood stream before it can be converted into cortisone by the oxidizing enzymes in the adrenal cortex. But under normal conditions or mild stimulation cortisone, leisurely elaborated from hydrocortisone, is secreted from the gland.

That the adrenal cortex should possess such adaptability to different demands and be able to respond efficiently but somewhat differently to normal or mild stimuli on the one hand, and to strong critical stimuli on the other seems reasonable—something like the production in peacetime of automobiles with chromium fixtures and white side-wall tires, and of equally efficient but more speedily assembled and less decorative cars in times of stress.

Investigators are waiting eagerly for enough hydrocortisone to see whether it will produce therapeutic effects that are better than or different from those of cortisone. Our own preliminary experiences (75) with hydrocortisone given intramuscularly or orally in a few cases of rheumatoid arthritis suggest that its antirheumatic and short-term metabolic effects are comparable to those of cortisone; certainly hydrocortisone acetate is not constantly or markedly superior to cortisone acetate; indeed in some cases it is inferior. Not enough long-term clinical or metabolic studies have yet been made to show whether or not free hydrocortisone or hydrocortisone acetate, long administered, can control rheumatic and other symptoms effectively but with minimal side effects.

*Metabolic Studies.*—Numerous metabolic studies have demonstrated the manifold effects which cortisone and cortico-

tropin have on the human economy: On the metabolism of fat, carbohydrates, protein and electrolytes, on certain enzyme systems, and so forth. (38, 47, 67, 68) By their great versatility and widespread influence cortisone and corticotropin rank as the most powerful hormones ever discovered. Although metabolic studies have not yet explained their mode of action, they have revealed the cause of some of the more important side effects, have helped us to define "overdosage" in biochemical as well as clinical terms and have provided certain means for the limited control or modification of side effects.

Cortisone has been labelled with tritium, a radioactive isotope of hydrogen (11); corticotropin has been tagged with radioiodine. (20, 63) From these new preparations much information on the metabolism of these hormones will be gained.

Studies on the short-term metabolic effects of free hydrocortisone or of hydrocortisone acetate given orally or intramuscularly have been reported recently. (14, 40, 66)

#### *Potentiation of Cortisone and Corticotropin*

In the hope of eliminating undesirable effects, attempts have been made to find some synergistic material which, when given with cortisone or corticotropin, would enhance the action of doses of the hormones which were too small to produce side effects. Salicylates, vitamin C, insulin and more recently sodium para-aminobenzoate have been recommended. (35, 71, 77) But among our rheumatoid patients no potentiation has resulted from these procedures.

#### *Diseases Which Are Responsive to Cortisone and Corticotropin*

Thanks largely to the pioneering work of Merck & Co., Inc. and of The Armour Laboratories, but also to the painstaking, frustrating, often thankless, but all-important work of a large number of trail-blazing physiologists and endocrinologists who during the past ten or fifteen years laid the foundation for the current clinical investigations, physicians have been able to test these hormones against a great variety of diseases.

*Diseases Usually Responsive.*—The effects of the hormones in some diseases are controversial, but a conservative estimate would indicate, I believe, that the symptoms of about twenty or twenty-five conditions, most of which were peculiarly resistant to previous agents, are usually and rather markedly suppressed by these hormones. Chief among them are certain rheumatic and articular conditions, acute or subacute inflammatory diseases of the anterior chamber of the eye, various allergic conditions including acute reactions of hypersensitivity to various pharmaceutical and other irritants (poison ivy, snake bites, certain insect bites), and sprue.

*Diseases Often Responsive.*—Less consistently and less strikingly affected than the conditions just mentioned are about twelve or fifteen in which results are often satisfactory, occasionally marked. Most of these conditions also have heretofore been very resistant to treatment. Among them are certain cases of acquired hemolytic anemia, chronic ulcerative colitis, regional enteritis, pemphigus, idiopathic hypoglycemia, early disseminated lupus erythematosus (also acute crises), periarteritis nodosa, and cranial arteritis.

#### *Relative Potency of Cortisone and Corticotropin*

If corticotropin, which presumably stimulates the production of at least three or four cortical hormones and not just cortisone, were found to be much better than cortisone for some diseases, or if corticotropin suppressed markedly certain diseases which were relatively unaffected by cortisone, such evidence would provide important clues to the pathogenesis of these various conditions and suggest that cortisone played a unique role in some diseases and that some other, perhaps unidentified cortical hormone played a leading role in others. But in *general* the various diseases react about the same to either hormone provided the patient has responsive adrenal glands. Some investigators have favored one or the other hormone in certain conditions (for example, gouty arthritis, asthma). But the differences may be due to other factors, such as unmeasurable differences in doses, since one cannot tell accurately *in any given case* how many milligrams of en-

dogenous cortisone are stimulated by a certain dose of corticotropin.

A discussion of the relative merits of cortisone and corticotropin would more appropriately concern the comparable cost, the differing ease of administration of one or the other, and their differing histopathologic effects on the adrenal cortex (stimulation and hypertrophy from corticotropin, suppression and atrophy from cortisone).

It is most fortunate that both are available; much clinico-physiologic information may be obtained by using each at different times in the same patient.

#### *Three Basic Plans of Hormonal Treatment*

Although there are many variations, there are three main plans of administration. (34) For acute or subacute self-limiting conditions the best plan is to give a single, relatively short course of one or the other hormone for a few days or a few weeks, daily doses often being fairly generous (large enough to suppress the acute inflammation) since prolonged usage is not intended or generally required. This plan is suited to such conditions as acute rheumatic fever, acute gouty arthritis, acute allergic reactions to drugs and so forth, cranial arteritis, and certain acute ocular conditions including sympathetic ophthalmia.

For chronic diseases there are alternative plans: the hormones are usually given either interruptedly in repeated courses, or for a prolonged period of time more or less continuously. These schemes are used for chronic diseases such as rheumatoid arthritis, chronic ulcerative colitis, early disseminated lupus erythematosus, and periarthritis nodosa. The plans for such chronic conditions will be discussed later.

*General Policy Regarding Dosage.*—Regardless of which plan is used the initial, higher suppressive doses should be gradually reduced, as soon as possible, to lower "maintenance doses."

#### *Undesirable Physiologic Effects: Side Effects*

*Relation to Dosage.*—Side effects are much less troublesome

and less frequent when a large amount (for example, 200 to 500 mg. or more of cortisone) is given as a single dose or when fairly large doses (100 to 200 mg. of cortisone) are given for only a few days or weeks than when lesser doses are given for a relatively long time. Side effects are becoming less troublesome as the newer conservative, low-dose schedules are being used.

Only a few side effects present emergencies; if side effects develop at all most of them develop fairly slowly, and can usually be detected early by the alert physician. Except for such things as cardiac decompensation, impending or obvious psychotic reaction, or significant aggravation of peptic ulcer, most of the side effects can be allowed to continue for a time if it seems desirable to adjust the doses with deliberation. The side effects are transient and "reversible" but since some of them present hazards, physicians who use these hormones should become familiar with the optimal methods of administration and with the procedures which help to prevent or control the side effects. (22, 32, 68) These have been described elsewhere. The best procedure is to prevent significant side effects by the use of the safer dose schedules. (4, 5, 75)

Experienced physicians still respect, but no longer fear, these side effects.

*Contraindications to the Use  
of Cortisone and Corticotropin*

I regard the following as absolute contraindications: psychosis, acute poliomyelitis, Cushing's disease, active tuberculosis, and possibly also early pregnancy and clinical amyloidosis. The other contraindications, which doubtless you have often seen listed, are relative ones which must be weighed against the patient's need for the hormone.

*Recent Experiences With the Two  
Great Rheumatic Diseases*

Most life insurance companies are now concerned directly or indirectly with health and disability insurance and with

diseases which promote chronic disability, loss of wages and the need for disability pensions. Therefore, the directors and medical officers of our great life insurance companies are trying not only to increase the span of life but to improve the quality of life. The support which your companies have given and can give to such organizations as the American Rheumatism Association, the American Heart Association and the Arthritis and Rheumatism Foundation is well placed and represents not only humanitarian cooperation but enlightened self-interest.

You have a great stake in the campaigns against the great killer, rheumatic fever, and against the great crippler, rheumatoid arthritis. Let me discuss the influence of the new hormones on these two main rheumatic diseases.

#### *1. Current Appraisal of the Hormonal Treatment of Rheumatic Fever*

Reports (8, 17, 53, 54, 78) on the effects of cortisone and corticotropin on rheumatic fever and its "complications" may appear to be contradictory and confusing unless they are broken down and studied in relation to each of the chief components of the disease. The problem of rheumatic fever must be separated into its four main parts: 1. the problem of the acute phase, the acute systemic disease; 2. that of acute rheumatic heart disease, myocardial and valvular; 3. that of chronic rheumatic heart disease, valvular and myocardial; and 4. the problem of prophylaxis, the prevention of recurrent attacks.

*The Acute Phase of Rheumatic Fever.*—Our further experiences with cortisone and corticotropin during the acute phase of rheumatic fever have just been reported in detail. (3) In our opinion the hormones are more rapidly and completely effective than salicylates. But we are not certain that they shorten the inherent duration of the acute rheumatic state. Therefore, hormonal usage must be continued until persistently negative clinical and laboratory data seem to indicate that the acute rheumatic state may have passed; this is done not only to prevent relapses from prematurely discontinued

treatment but in an attempt to suppress fully the acute cardiac lesions which may induce chronic carditis.

*Acute Rheumatic Carditis.*—It seems to us likely that the hormones do suppress acute carditis, at least in some cases, because certain diastolic and systolic murmurs have changed or disappeared; also dyspnea, orthopnea, cyanosis, gallop rhythm and signs of pericarditis have diminished or disappeared. In some of our cases enlarged cardiac profiles decreased notably and we believe that the reduction in the size of the cardiac shadow was not always due, as some believe, merely to diminution of pericardial fluid. Re-examination of the hearts several months later revealed no new or increased old carditis. But it will take several years before one can tell whether or to what extent these hormones can prevent the development of chronic carditis.

*Chronic Rheumatic Carditis.*—Old valvular lesions with their related murmurs, and old cardiac enlargements are not affected by the hormones, which cannot influence such irreversible lesions.

*Prophylaxis.*—The hormones have no prophylactic value against acute recurrences. Therefore, long-term daily chemoprophylaxis should be instituted whenever possible. (44, 52, 61, 74)

## 2. *Cortisone and Corticotropin for Rheumatoid Arthritis*

*Plans for Administration.*—The two basic plans for giving these hormones in cases of rheumatoid arthritis, or any other chronic disease, are 1. interrupted administration—that is, repeated courses of one or the other hormone are given, each course lasting a few weeks or months depending on the severity of the disease and the response to treatment; the courses are separated by periods without the hormones; and 2. prolonged administration—that is, the more or less continuous use of one or the other hormone for an indefinite time, or in other words, for as long as results are satisfactory and significant side effects do not develop.

Several variations of each of these two plans have been used; each modification has some refinement or supposed

advantage. (34) I shall discuss here, however, only the two basic plans.

Physicians should know the purposes, advantages and disadvantages of each plan (and its modifications) and should explain them simply but carefully to each patient before treatment is begun. The plan should be chosen to fit the medical requirements, finances and psychology of each patient.

*Purposes, Advantages and Disadvantages of Plan 1.*—The purposes and advantages of plan 1 (interrupted administration; repeated courses) are 1. to lessen the incidence of the ordinary side effects: patients so managed would have few major side effects; 2. to see whether a posthormonal remission of significant length will develop without the need for prolonged, indefinite treatment; such remissions develop in about 10 per cent of patients; 3. to interfere as little as possible with the patient's pituitary-adrenal mechanism in deference to the hypothetical development of some long-range side effects of a type not yet encountered; and 4. to minimize the cost of the hormones.

The disadvantages of plan 1 are 1. the relapses between courses, 2. the common inability of the patient to readjust himself to the return of his symptoms even though he knows the relapse can be controlled by the next course, and 3. the possibility of a "rebound attack" in which the patient feels worse than before the course was started. True "rebounds" are not common in our experience; most of them can, I believe, be prevented by ending the courses gradually, that is, by tapering off the doses over the last two or three weeks, rather than stopping the course suddenly. Many so-called rebounds are probably not flares due to posthormonal adrenocortical insufficiency, but are symptomatic reflections of a disease which worsened even while its manifestations were being hormonally suppressed.

Our own results from this plan have been described in detail (32): although symptoms were markedly relieved, the incidence of prolonged posthormonal remissions was low.

Patients satisfied with this program are those whose relapses, after treatment is stopped, are tardy and incomplete, or those who, concerned about long-continued treatment, prefer, despite relapses, to give their "glandular system" a chance to recover its "physiologic equilibrium."

*Purposes and Disadvantages of Plan 2.*—The purpose of prolonged administration is, of course, to provide as much relief for as long as possible without significant side effects or relapses. The disadvantages of this plan are 1. the cost of continued medication, 2. increased liability to the ordinary side effects if relatively high maintenance doses are required, 3. hazards, poorly defined as yet, related to prolonged "interference" with the pituitary-adrenal system (possibility of unsuspected intercurrent infections masked by the hormones; uncertain results from the prolonged cortical adrenal atrophy induced by cortisone or from the hypertrophy induced by corticotropin), and 4. the lack of opportunity, which a "rest period" affords, to see whether a spontaneous or induced remission may have developed.

Holding out hope for prolonged relief, this plan has a strong appeal to physicians and patients. It has been made feasible by the introduction of cortisone tablets which are of course easier to use continuously than injections of corticotropin. In the state of our present incomplete knowledge the plan of prolonged administration involves more of a calculated risk than does plan 1. But, aside from the fact that "spontaneous remissions" might not be recognized readily, plan 2 appears to be preferable, certainly for patients who do well on doses nonproductive of side effects.

Later herein I shall review briefly our recent experiences with this plan.

*Common Reasons for Unsatisfactory Results.*—Among patients treated by us or by others we have noted certain common causes for an unsatisfactory result:

1. Too rapid reduction of dosage; this is perhaps the commonest cause. We have the dubious distinction of having

been the first to make this error. (30, 32) In our earliest (severe) cases flares commonly developed when doses were reduced from 100 to 75 mg. or from 100 to 50 mg. of cortisone. Many patients do well until the daily dose of cortisone is reduced from 100 mg. to 50 mg. directly instead of gradually; an example of gradual reduction would be from 100 mg. to 87.5 mg. daily for a few days, then to 75, then to 62.5 and later to 50 mg.

Cortisone tablets are fairly small; a half-tablet (12.5 mg.) looks quite insignificant but that unimpressive half-tablet alone contains enough to keep most addisonian patients in daily cortisone equilibrium. The importance of slow, gradual reductions in dosage and the fact that there is a real difference between, for example, daily doses of 50 and 62.5 mg. need greater emphasis and wider appreciation.

2. Erratic dosage. Some patients are receiving erratic, vascillating dosages. For example, one disappointed rheumatoid patient came to us after this experience: he did well on cortisone given at the rate of 100 mg. daily for two weeks, then his joints flared when the dose was suddenly dropped to 50 mg.; to "recover lost ground" he was then given 150 mg. daily but side effects began to develop; although they were minor, the dose was reduced, not gradually as it should have been, but precipitously to 50 mg. daily for a few days, then to 25 mg. when the side effects did not completely disappear immediately; as a result of such erratic dosage the joints flared and the use of the hormone was discontinued. This "course" was like that of an automobile veering rapidly from side to side.

3. Insufficient dosage. I have seen several patients who have been given injections of corticotropin never more than once a day, or only once or twice a week, or who have never received more than 25 mg. of cortisone daily or every three to seven days.

4. Neglect of measures to control side effects. Although side effects are prevented or controlled best by careful attention to dosage, other measures are helpful and important. (22,

32, 34, 68) Some conservative, overly cautious physicians prematurely abandon effective moderate-sized doses when mild or moderate side effects develop (for example, mild pretibial edema) without first attempting to continue the (temporarily required) doses by adding suitable controls (salt restrictions; use of mercurial diuretics, potassium salts, estrogen, and so forth). Sometimes when the latter measures are used temporarily and the dosage of cortisone is reduced slightly, the side effects are controlled and may not reappear when the dosage is increased again.

5. Failure to use "booster doses." The notorious capriciousness and unpredictable variations in the activity of rheumatoid arthritis are its chief characteristics. In precortisone days major exacerbations, presumably due to increased activity of the unknown etiologic agent, were common and were simply taken for granted; they were either accepted and "seen through" sadly but philosophically or they occasioned a short flurry of physical therapy, doubled doses of aspirin or a visit to a spa. But nowadays when flares (unexpectedly) develop in a patient whose symptoms have been satisfactorily controlled by hormones for some weeks or months, the flares appear to be "mysterious," and are commonly blamed on the patient's "improper reaction" to the hormone rather than on the disease itself. Such flares disappoint all concerned, especially the patient who has not been forewarned that they may occur despite considerable hormonal protection. The new hormones, especially in the low maintenance doses now being used, do not prevent or adequately conceal *major* flares. At any time the disease may increase in activity and may break through the hormonal "protection." Unlike many diabetic patients whose hormone deficit and insulin requirement may remain relatively constant for long periods (barring special stresses), many rheumatoid patients require frequent adjustments (increases or decreases) in the daily dosages of cortisone or corticotropin as their underlying disease waxes and wanes.

Rheumatoid patients receiving cortisone or corticotropin may note minor flares lasting one or two days a week or for

a few days every now and then. This "arthritis in miniature" is common and usually needs little or no adjustment of dosage. But more marked flares require a temporary increase of the daily dose ("booster doses"), sometimes for only a few days, in some cases for several weeks. (6, 7, 75)

6. Physical irritation. As soon as their symptoms begin to abate under treatment, many rheumatoid patients forget that their underlying disease may be essentially unchanged. Delighted by the lessening of pain they prematurely and sometimes rather disastrously re-engage in strenuous physical activities which they have not been able to do for months. It is not uncommon to find rheumatoid patients, after a few days of hormonal treatment, walking a couple of miles at a stretch, something that some of them did not do even in health. I have noted several patients who arrived in Rochester rather disabled but who later (within two or three weeks after starting treatment) "passed the time" by playing nine to eighteen holes of golf daily. We must caution each patient to increase his physical activity only gradually and to continue to avoid irritating trauma despite the hormonal suppression of symptoms.

7. Other reasons. There are other rare or hypothetical factors which have been suggested as being responsible for a loss of relief from doses which were previously adequate, among them being a gradual tolerance to any one-species brand of corticotropin (a change from corticotropin derived from hogs, for example, to that from horses or cattle often corrects matters) and the occasional development of "corticogenic hypothyroidism." (22, 79) (Although small doses of thyroid are said to restore the effectiveness of the cortisone or corticotropin, our results from the additional use of thyroid have been disappointing.)

The reasons for the loss of hormonal relief in some cases are as yet quite unknown.

#### *Our Recent Results From Cortisone Given Continuously*

Last week we reported our results from cortisone given daily, usually by mouth, to 100 rheumatoid patients for many

months (up to twelve months). (75) Symptoms were reduced markedly or very markedly in 65 of the 100 patients, moderately in the rest. Expecting to give these patients cortisone (tablets) indefinitely, we commonly administered suppressive doses which were smaller than those we had given previously by injection. Initial suppression of symptoms was accomplished in 31 per cent of cases by doses of 75 mg. or less given daily. Furthermore, suppression was commonly maintained (in 71 per cent of cases) by the use of maintenance doses of only 62.5, 50, 37.5 or 25 mg. a day.

As stated above, one of the most important factors leading to the satisfactory, continuing control of symptoms was the gradual reduction in dosage from the early suppressive doses to the low maintenance doses. The daily dose was reduced by only 12.5 to 25 mg. at intervals of two to seven days or more, depending on the patient's clinical response.

In none of the 100 cases have side effects necessitated discontinuance of treatment. No side effects have developed in about half of the patients; most of those which developed in the other half were mild. Side effects occurred in 63 per cent of patients receiving 75 mg. or more daily but in only 21 per cent of those receiving less than 75 mg. daily.

These results indicate that the oral administration of cortisone in small doses, short of significant side effects, is useful for the long-term "investigative management" or treatment of many patients with this disease.

#### *Hormones Compared to Other Remedies for Rheumatoid Arthritis*

How do our results from the use of cortisone compare with those from more orthodox measures? Although some patients obtain satisfactory results from a simple program of "general measures" (aspirin, physical therapy, nutritious diet and sufficient reduction of physical activity to avoid irritating trauma), far too many do not. They then have a choice of chrysotherapy, treatment with hormones, or self-neglect. Chrysotherapy is cheaper, and when results are obtained from

treatment they may last longer than those from treatment with hormones. However, marked relief (a more or less complete symptomatic remission) results from chrysotherapy in only about 15 per cent of cases (27), while from the hormones such relief is obtained much more often (in about 60 to 65 per cent of cases). Minor undesirable effects result about as often from the use of gold as from the use of cortisone; the troublesome effects of the hormones are certainly no more so than those from gold.

Some physicians, disappointed because at this early stage of our knowledge "safe doses" of the new hormones often cannot provide maximal relief (as compared to an optimal or submaximal effect), recall with surprising nostalgia the virtues of chrysotherapy. (12) I cannot agree with them. In my opinion their enthusiasm for "the good old days" is misplaced. Although chrysotherapy helps some patients and is still useful (9, 69), it is no better in 1951 than it was in 1947. (27)

Some rheumatologists have combined chrysotherapy with hormone therapy. (23, 39, 48) But neither appears to potentiate the other. Of course if chrysotherapy seems indicated, in selected cases cortisone can be used during the early, otherwise difficult weeks or months of chrysotherapy in order to control the rheumatic symptoms until the effects of gold salts begin to appear—if they do appear.

Something is sorely needed for the countless thousands of rheumatoid patients whose disease is not materially or adequately controlled by "general measures" or by chrysotherapy. For patients who need more relief than safe doses of cortisone or corticotropin can supply by our present methods of administration, the best plan is to combine the use of one or the other hormone with "general measures"; aspirin, physical therapy, moderation of physical activity. Such a combined program is, in our opinion, often the treatment of choice.

#### *Adventures Ahead*

For us rheumatologists there are interesting times ahead. Intriguing and stimulating pieces of information are being

brought to our attention, pieces of an exciting jig-saw puzzle that will be put together in time.

We are told that there appears to be a histologic lesion in the pituitary gland which has been found so far only in patients who have had either rheumatoid arthritis or Addison's disease. (58) We are told that a strange adrenocortical metabolite has been found in the urine of several rheumatoid patients and that rheumatoid patients (16) do not metabolize the steroid, progesterone, normally. (62) These reports must be checked and rechecked.

The antirheumatic substance X of pregnancy may or may not be cortisone (or hydrocortisone). (1, 16, 26, 51, 73, 80) Substance X may not be one substance but more than one, or one chief substance modified by the special circumstances characteristic of pregnancy. Cortisone (or hydrocortisone) is, to date, the most likely contender for the position of substance X. But if cortisone is substance X, how do pregnant rheumatoid (or nonrheumatic) women handle their markedly increased, antirheumatic amounts during the several months of pregnancy in a manner so that significant side effects do not develop? During pregnancy, or otherwise, does some additional hormone, a regulatory one, or some regulatory, protective mechanism cooperate to keep the markedly anti-rheumatic quantities of cortisone clinically effective but non-productive of side effects? When we learn how the pregnant rheumatoid patient utilizes her cortisone-like hormones, then perhaps we can give at will to most rheumatoid patients complete relief for months on end without the development of significant hypercorticism.

If, as some believe, the antirheumatic substance X of jaundice is not cortisone (15, 19, 46, 66), or at least does not depend on increased production of cortisone, either it is an antirheumatic substance just as good as cortisone, or some cooperating agent or mechanism operates temporarily to make "normal" amounts of cortisone mysteriously effective. Either possibility is an arresting one. We must study with greater care than ever these two potentially revealing phenomena,

the mechanisms of relief of certain diseases by pregnancy or jaundice.

After last week's world series baseball games here in New York it seems appropriate for me to use the language of baseball in closing. Three short years ago I remarked that I thought we were on "first base" so far as a control for rheumatoid arthritis was concerned. Now I believe we are nearing second base.

With these new hormones as research tools we can face the adventures of the future with much confidence and hope.

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## CORTISONE AND HYDROCORTISONE 33

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PRESIDENT YLVISAKER—Dr. Hench, we cannot thank you enough for this wonderful talk and demonstration. I assure you that it is a source of great satisfaction to all of us to know how much you are doing for all these people.

Those of you who heard Dr. Dublin speak the other day on the course of life insurance medicine heard him tell about the wonderful progress that has been made in the control of all infectious diseases. He told us that many of these infectious diseases have been eliminated altogether. Others have been brought under control and leave very few residuals for us to worry about.

In our own company today only 5 per cent of all deaths are due to infectious diseases. Not long ago several of the infectious diseases were the leading causes of death. We still have to contend with tuberculosis and rheumatic fever. We know that tuberculosis is on its way out. Not many years ago there were some 53,000 deaths a year from tuberculosis. Then it went down the next year to 48,000, the next year to 43,000, the next year to 40,000. Last year there were only 33,000 deaths from tuberculosis. We hope for complete elimination, and I hope to live long enough to see tuberculosis as rare as typhoid fever is today.

The question of rheumatic fever then arises. We have heard from Dr. Hench about the wonderful effects of these hormones on rheumatic fever, and all of us are wondering what the result will be. Will our infectious disease control and our treatment by these hormones help us to eliminate rheumatic fever from our problems?

We still have many problems with applicants who come to us with a history of rheumatic fever, with fully developed rheumatic lesions, and with systolic murmurs that seem to be residual from rheumatic fever.

In order to bring this discussion before you, we have asked one of our youngest members — in fact he was admitted here only today. I happened to meet Dr. Howard M. McCue, Jr., at White Sulphur Springs a year ago last June, had dinner with him, and heard him discuss the work they were doing in Richmond, Virginia, on rheumatic fever. I felt that he could well represent our Association in discussing this problem before us.

Questions that we are going to ask him are: Will our present infectious disease control program, and will the administration of these hormones eventually help us to control rheumatic fever and residual rheumatic heart disease?

It is a pleasure to have you here, Dr. McCue. We want to welcome you to membership in the Association, and we are glad you have not lost time in appearing on our program.

## THE OUTLOOK FOR THE CONTROL OF RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

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Rheumatic fever and rheumatic heart disease are leading causes of death in children and young adults. Any serious disease affecting children always evokes considerable general interest. A prominent cause of mortality in a young age group is also of considerable interest to the medical profession, particularly to those members concerned with the evaluation of risks for life insurance.

Rheumatic fever is, from a practical standpoint, more of a clinical than a pathologic entity. In the absence of a single specific diagnostic test for the presence or absence of rheumatic activity, there must remain some disparity between the clinical and pathologic incidence. This fact obviously alters the accuracy of our statistical observations that follow clinical studies. The presence of clinical evidence of carditis clarifies the situation to the extent that there is usually little doubt of the presence of the rheumatic state in this group.

The etiology of rheumatic fever is not clear. The most important widely accepted concept has to do with the relationship of the disease to group A beta hemolytic streptococcal infection. It is generally felt that a definite relationship exists between such infection and the subsequent development of the connective tissue lesions of the rheumatic state. The mode of reaction and development of these granulomatous lesions remains unknown. An interesting concept is that the mechanism consists of the development in a susceptible host of autoantibodies. There is available experimental work to support such a contention. Nonetheless, we must still approach control of the disease clinically on an empirical rather than a rational basis.

The most reasonable and practical approach to control of rheumatic fever is through the attempt to control beta hemolytic streptococcal infections. Within the past fifteen years numerous antibiotic agents have been developed, and the overall effect on the streptococcus has been disastrous. Penicillin, judiciously employed, offers the most effective method of combating this infection. In 1949-50 a study conducted at an Air Force base in Wyoming employed penicillin in the treatment of exudative pharyngitis in 1178 patients; 1162 men (selected by the last digit of the serial number) served as untreated controls. Only two cases of definite acute rheumatic fever developed among the treated group in contrast to 28 cases among the controls. I would accept this as evidence of the efficacy of prompt treatment with penicillin in reducing the incidence of subsequent clinically recognizable rheumatic fever.

Once acute rheumatic fever has developed, effort should be directed toward management of the patient in the best available environment under careful medical observation. The most important single development in the field of active therapy occurred in 1949 when Drs. Hench, Kendall, Slocum and Polley first employed cortisone in the treatment of acute rheumatic fever. There is diversity of opinion as regards the results of treatment with ACTH or cortisone. The general consensus seems to be that if employed early in the acute phase these agents may shorten the course of active rheumatic fever and possibly reduce the hazard of permanent cardiac damage. Equally important is the fact that in some instances it seems to be a life-saving procedure in the critically ill patient with severe carditis. It is not felt that these hormones alter pre-existing valve deformity. These opinions coincide with the results we have observed in the treatment of 14 such patients by Dr. Carolyn McCue on the rheumatic fever ward at the Medical College of Virginia.

Approximately 65 per cent of a group of 1,000 children admitted between 1921-1931 to the House of the Good Samaritan in Boston with rheumatic fever developed some type of rheumatic heart disease. This figure is unquestion-

## THE CONTROL OF RHEUMATIC FEVER, ETC. 37

ably high in that these children were sufficiently ill with their initial illness to require hospitalization. I feel that a figure of 50 per cent would be more accurate for all cases of recognized rheumatic fever. The presence of a valvular lesion subsequent to the cessation of rheumatic activity presents a somewhat different problem from that of the acute illness. There must be rehabilitation, training, social assistance where needed, and careful follow-up observation, but primary in importance is the prevention of recurrence of active rheumatic infection. Many measures have been and are still employed: transfer to a low incidence geographic area, careful attention to environmental, nutritional and other factors of general resistance. By far the most practicable and efficacious measure is prophylaxis against streptococcal infection with oral antibiotics. Sulfonamides have been most widely used and reports are generally good. The recurrence rate in short term studies is usually 3 to 5 times greater in the control than in the protected group. The obvious defect is the subsequent development of sulfonamide-resistant strains of streptococci. Toxic reactions to the drugs occur and must be watched for and the drug promptly withdrawn if they develop.

We are in the midst of a study using Gantrisin® in the Rheumatic Fever Clinic of the Medical College of Virginia. Results have not been tabulated, but our current impression is favorable. We must admit that toxic reactions are by no means a rarity. Of greater prophylactic effectiveness, with less risk, is the use of oral penicillin. As generally employed, the cost of such treatment averages \$9.00 to \$10.00 per month and this is at present the greatest barrier to its widespread use. Reports of studies employing penicillin as the prophylactic agent are even more glowing than those of the sulfonamides. In a large study of a special group of Chicago school children given 800,000 units daily for the first week of each month of the school year, the recurrence rate of rheumatic fever was zero in the treated group compared to 11 per cent

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and 19 per cent in two control groups followed during one complete rheumatic fever season.

Equally as important as an organized program of prophylaxis is the prompt institution of adequate antibiotic therapy for any infection that occurs in the patient with rheumatic heart disease. Upper respiratory and other related infections should be promptly treated with penicillin in adequate amounts and over a sufficiently long period of time to completely eradicate the possibility of further activity of streptococci. Such results promptly produced drastically reduce the chance of reactivation of rheumatic fever.

Subacute bacterial endocarditis is a statistically small but potential hazard in the presence of a rheumatic valvular lesion. In the group followed by Jones, Bland and others in Boston over a twenty-year period, it accounted for 10 per cent of the deaths. For the past eight or nine years there has been a radical change in this aspect of the disease as a result of the remarkable effectiveness of penicillin, streptomycin, aureomycin and other related agents in the treatment of this infection. In contrast to a previous mortality of almost 100 per cent we now rescue something over 80 per cent of patients with bacterial endocarditis.

As years pass, the majority of patients with established rheumatic heart disease eventually encounter cardiac difficulty. A substantial percentage who go through a reasonable period of growth without recurrence of rheumatic fever will have considerable reduction or even complete loss of the clinical manifestations of the valvular lesion. This group is approximately balanced by those who, having only potential rheumatic heart disease at the cessation of active rheumatic fever, ultimately are found to have a serious valvular lesion. At present we are better able to manage the major complication, congestive heart failure, than we were some years ago. Digitalis glycosides, improved mercurial diuretics and the more vigorous employment of them, procaine amides, intramuscular quinidine preparations, positive pressure oxygen administration, and control of electrolyte balance have all improved the management of this con-

## THE CONTROL OF RHEUMATIC FEVER, ETC. 39

dition. The obvious result of such progress is to at least delay the principal mechanism of death in rheumatic heart disease.

The serious mechanical defect of mitral stenosis, particularly in those patients with approximately normal sized hearts and reasonably good function between bouts of acute pulmonary edema, has tempted the surgeons for a good many years. Cutler, Souttar, and others made an abortive effort in this direction a number of years ago. As a result of the combined efforts of Smithy, Harken, Bailey, Blalock, and others, plus the improved surgical and anesthetic technics and technics of physiologic study, notably cardiac catheterization, the first reports of really successful valve surgery appeared in 1948. At present several hundred cases of mitral stenosis have been subjected to valvotomy with generally favorable results. The operative mortality in reported series has varied from 15 to 25 per cent and with careful selection of cases it is steadily improving. The percentage of favorable results is high—approximately 75 per cent of those surviving the procedure.

I have attempted to review those aspects of rheumatic fever and rheumatic heart disease which are important in the control of the disease. It is my impression that the outlook for control is reasonably good. At the present time, penicillin and other antibiotic agents are so readily prescribed for any type of infection as to substantially reduce the threat of hemolytic streptococcal infection in that segment of the population that seeks medical care. In addition, carefully organized prophylaxis has certainly reduced the recurrence rate of active rheumatic infection. Accurate early diagnosis and wise management, including the judicious use of ACTH and cortisone, improve the results in the acute phase of the illness. Long term followup under good medical care has improved the outlook for those having rheumatic heart lesions after the active stage. Subacute bacterial endocarditis has all but succumbed to the antibiotics. Congestive failure is better managed than it once was, and most dramatically the surgeon has attacked the most serious valvular lesion

resulting from rheumatic infection, tight mitral stenosis. All of these steps along the path of medical progress have contributed to improvement of the outlook for the potential or actual rheumatic fever patient.

Reports of mortality trends and follow-up studies confirm the fact that the situation has improved. It appears from the work of Jones, Bland, Wilson, Ash, Martin and others that the greatest mortality occurs in the first year following the acute illness. After this the situation gradually improves until age 18 to 20, and past this age the mortality hazard becomes remarkably reduced. In 15 to 20 year follow-up studies approximately 40 to 45 per cent of deaths occur within 5 years of the initial infection and 80 to 85 per cent of the deaths occur prior to age 20. Among white industrial policy-holders of the Metropolitan Life Insurance Company aged 5 to 24, deaths from rheumatic fever and rheumatic heart disease have declined approximately 80 per cent since 1920; by far the greater part of this reduction has occurred in the past decade.

From the practical underwriting standpoint, the improving situation plus the improvement in medical examinations, the availability of specially trained examiners, and the increased use of special studies has in recent years resulted in our insuring individuals impaired by rheumatic heart disease who would formerly have been rejected. Many factors must be considered in evaluating such a risk; heart size is the most important single observation. The presence of considerable cardiac enlargement and history of congestive heart failure are very unfavorable findings. The specific valvular lesion must be taken into consideration. Mitral stenosis is the worst lesion from a prognostic standpoint, and aortic insufficiency is less favorable than mitral insufficiency. The presence of auricular fibrillation is so unfavorable as to be unacceptable. And lastly there should be a general estimate of cardiac function.

It is perfectly obvious that these cases are not standard risks under any circumstances; but the progress in knowl-

## THE CONTROL OF RHEUMATIC FEVER, ETC. 41

edge of the natural history of the disease has provided us a working basis for evaluation, and the improvement in prognosis in recent decades leads me to believe that with careful selection we have every reason to feel that acceptance of such cases is sound medical underwriting.

**PRESIDENT YLVISAKER**—As you know, Dr. McCue is associated with Dr. Ennion Williams and Dr. G. M. Harwood in the medical department of the Life Insurance Company of Virginia. I have asked Dr. Williams to discuss this paper.

**DR. WILLIAMS**—Improvement in mortality affects selection for life insurance in proportion to the chronicity of the disease involved. The death rate from pneumonia and influenza has declined 88 per cent during the past 25 years. This has been very satisfying but it has had *no effect* on the selection of applicants with a history of pneumonia. The prognosis of syphilis, diabetes and pernicious anemia has improved. This improvement *has* permitted a more liberal policy for granting life insurance to persons with these diseases.

Rheumatic fever falls in between these two categories. Dr. McCue has presented, very clearly, progress in the management of this disease which has resulted in a lessening in the incidence of the disease, control of the acute rheumatic state, and improved treatment of rheumatic heart disease. Our ratings for persons with a history of rheumatic fever or with evidence of rheumatic heart disease cannot be reduced in proportion to this improved prognosis. Our ratings are based on the ratio of the mortality of the impaired group to the mortality of the general population. We must, therefore, compare the improved outlook in rheumatic fever with the improvement in overall mortality.

Last month Dr. Dublin reported an exhaustive study on health progress before the Society of Actuaries. His figures indicate that the death rate among white men for all causes for the age group 15 to 24 declined 62 per cent between the years 1926-30 and 1950. During this period, for the same age groups, the death rate from cardiovascular renal diseases

declined 69 per cent. For white women the death rate from all causes declined somewhat faster than the death rate from cardiovascular disease.

In this age group, 15 to 24, it may be assumed that the heart disease is chiefly rheumatic heart disease. These figures would, therefore, suggest that improvement in mortality from rheumatic heart disease has approximated the improvement in mortality from all causes. Rheumatic heart disease maintains about its same relative importance as an impairment. Heart disease still ranks third as a cause of death between the ages 5-24. It is important, therefore, to keep in mind the factors affecting prognosis in rheumatic fever and rheumatic heart disease.

Dr. McCue has outlined the factors we consider important: the age of the patient, the frequency and severity of recurrences of rheumatic activity, the type of valvular lesion, heart size, evidence of congestive failure, evidence of pericarditis or of abnormal rhythm, and the existence of impaired exercise tolerance.

PRESIDENT YLVISAKER—Thank you, Dr. Williams.

We have asked one of our Canadian medical officers to present the next number on our program. Our Canadian friends have contributed much to this organization. We are missing several of them today. Dr. Samuel J. Streight retired last year and wrote me a letter requesting me to bring you all his greetings. We also miss Dr. Birchard who served for many years on our X-ray and Electrocardiographic Committee. He seemed in his usual health when we saw him last at the meeting in June at Colorado Springs. We were shocked to hear of his sudden death in July.

Dr. J. Gilbert Falconer of the North American Life Assurance Company of Toronto, has reviewed recent developments in the studies of blood dyscrasias and will discuss the relation of these developments to our underwriting problems. Dr. Falconer—“Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis.”

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: THE EFFECT OF TREATMENT ON PROGNOSIS

J. GILBERT FALCONER, M. D., M. R. C. P.

*Medical Referee*

*North American Life Assurance Company,  
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This paper is an attempt to review and summarize the literature of recent years in relation to the blood dyscrasias. You are all familiar in a general way at least with the material which I have collected. I have no mortality study to present. Any suggestions which I shall make from the point of view of life insurance medicine are entirely the result of my own thoughts on these subjects. You will no doubt have your own opinions as to the effect of these diseases on longevity, and I shall be very glad indeed to receive your criticisms.

### *PERNICIOUS ANEMIA*

The most important of the blood dyscrasias from the point of view of the life insurance companies is undoubtedly pernicious anemia. This disease does not appear to have received any particular attention from the insurance world, and I can find no definite study of it in the insurance literature, with one minor exception.

Various statistics in the United States and Canada show that there are probably somewhere between seven and nine cases per hundred thousand people, and some authors estimate that there may be as many as thirty-five per hundred thousand people in some areas. The number seems to be slowly increasing, apparently due to more accurate diagnosis and also to the generally increased longevity. The cases are reported mostly between the ages of 45 and 70. When one adjusts for the number of living people in each decade, it is quite obvious that there is a steady increase as each group in the population becomes older. Cases appearing in the 20's and 30's are on the whole medical curiosities and will not often be presented for insurance. Even in the early part of the fifth decade not many are discovered.

It is stated by Dr. William P. Murphy in his book "Anemia in Practice", that it should be possible to eliminate death from pernicious anemia as a result of early and proper treatment. However, it is impossible to attain this happy situation in spite of the fact that we have a specific substance available because unfortunately one cannot hope to obtain the uniformly intelligent use of this material. The Metropolitan Life Insurance Company (1) published some figures after following a group of insured lives from 1921 to 1935. This showed a gradual decline in the death rate from pernicious anemia after 1926, and in 1935 the death rate was about 40 per cent of what it had been ten years previously. The Vital Statistics Division of the U. S. Department of Commerce shows the death rate varying from 5.3 to 5.8 per hundred thousand before 1926 and a death rate of 2.3 per hundred thousand in 1935. The figures available in the medical literature in regard to the death rate from pernicious anemia undoubtedly show a gradual decline but unfortunately they are not such as can be used to form any sort of accurate prediction as to true mortality rates. Perhaps something of this type will be available before very long.

In regard to possible increases in the mortality rate of groups who suffer from pernicious anemia, one must consider several causes. First of all, the matter of accurate diagnosis. There are other diseases which cause hyperchromic, macrocytic anemia such as short circuiting operations on the stomach, intestinal strictures, cirrhosis of the liver, extensive carcinoma of the stomach, sprue, myxedema, subleukemia, nutritional anemia, pregnancy, nephritis and *Diphyllobothrium latum* infestation.

There also seem to be cases of chronic ill health associated with mild anemia, the causes of which are not clear and which are frequently diagnosed as pernicious anemia by reasonably competent physicians. In doubtful cases it might be well to inquire specifically for the results of such things as the histamine test in regard to gastric achlorhydria and the reticulocyte response if this has been observed. As a double check on the accuracy of the diagnosis, I would suggest that we wait until the applicant has been under treatment for one full year. By

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: 45

the end of that time many of the doubtful cases will be eliminated from the group.

Having assumed that the diagnosis is accurate, one must consider next the matter of the adequacy of treatment and the cooperation of the patient as suggested in the physician's report. Patients should be receiving at least 15 U.S.P. units of liver extract every three weeks. If there has been any evidence of spinal cord involvement, they should be receiving at least four times this amount.

Vitamin B12 has received a great deal of attention and is now replacing liver extract in the hands of a great many physicians. This is very natural in that it is a pure product and seems to be a specific substance. In switching over to vitamin B12 from liver extract it has been felt that one microgram of the vitamin was approximately equal to one U.S.P. unit of liver extract. This is, however, very inconstant from the practical point of view. It has been found that patients on the uniform dose of about thirty units of liver extract every three weeks would require anywhere from thirty micrograms of B12 every four weeks to one hundred micrograms every two weeks. In other words, when the switch-over is made each individual patient must be reassessed in regard to his reaction to vitamin B12. It is undoubtedly true that some patients can be maintained in good health with smaller and less frequent dosage when liver extract is used. It seems that for treating the acute state with or without cord involvement and for maintenance therapy, vitamin B12 is thoroughly efficient if used in adequate amounts.

Folic acid was a very great assistance to those who became sensitive to liver extract. It was able to control the anemia quite readily. Unfortunately cord degeneration not infrequently developed in spite of the control of the blood picture by folic acid. An occasional case of pernicious anemia will not respond properly to liver extract or vitamin B12. Folic acid will usually bring satisfactory results in these people. Under the latter circumstances the patient may appear to be perfectly well, but one must remember the tendency to develop subacute combined degeneration.

A poorly treated patient will show increased mortality partly as a result of complications in other organs resulting from mild uncorrected anemia. The heart muscle of an older patient with only moderate coronary sclerosis may show the signs of suffering from anoxemia as a result of inadequate treatment of his pernicious anemia. This may be seen as congestive failure or angina pectoris.

After establishing the accuracy of the diagnosis, the degree of cooperation on the part of the patient, and the adequacy of treatment, there are still conditions frequently associated with pernicious anemia which might cause an increased mortality rate. The greater the degree of neurological involvement before the commencement of treatment, the less likely is one to find complete recovery. I suggest that cases with some posterior column degeneration as manifested by numbness and tingling of the feet and hands can be accepted as ordinary cases of pernicious anemia. Cases which also show lateral column disease should be selected with much more care and should be considered to be a much less desirable group. If the cord degeneration has advanced so that a condition of "cord bladder" is present, the outlook is serious. They rarely show much improvement under treatment, and the urological infection that so often accompanies a "cord bladder" will interfere greatly with the efficiency of the anti-anemic treatment. With a slight lapse in treatment this last group will rapidly develop serious neurological signs and symptoms again, leading to permanent disability or death in spite of treatment. On rare occasions patients with pernicious anemia present themselves with only a mild degree of anemia but with rather severe cerebral disturbances. These patients are not infrequently suicidal and their prognosis as a rule is not good.

It has been stated frequently that gallbladder disease is often associated with pernicious anemia. In a very recent paper, Cameron, Townsend and Mills (2), of Montreal, summarize the results of their observations over a period of twenty years. They state that more than half of the patients originally presented themselves with gastrointestinal symptoms and

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: 47

that at the end of their period of observation more than half of these still had gastrointestinal symptoms. Whether or not these symptoms were due to recognizable organic disease of the gastrointestinal tract is not stated in the article. Gastric carcinoma has often been held to be an important cause of death in pernicious anemia. A considerable number of authors do not agree with this statement. From a perusal of the literature I would conclude that pernicious anemia cases are somewhat more likely to develop gastric carcinoma than are normal people.

Many years ago it was stated that cases receiving liver therapy frequently developed hypertension. This aspect has not been considered very often in the literature, but in the paper which I mentioned above the authors note that at the beginning of their observation only two out of fifty-one had blood pressures of above 145/90, whereas at the end of their observation forty-seven out of a total of sixty-three showed readings above that level. In twenty-six cases the diastolic pressure was 100 or higher by the end of the period of observation. Although many of these patients had become quite old, one cannot accept entirely the statement that this was merely due to their advancing ages. On the other hand it is interesting to note that in the same article the authors state that no cases of malignant hypertension were seen, nor were any of the patients incapacitated because of symptoms attributable to hypertension. I draw no final conclusions about the matter of associated gastrointestinal disease or hypertension but I think that these factors should be drawn to your attention.

When all these aspects which may have a bearing on the ultimate mortality are considered, I would feel that one must walk warily from the insurance point of view. In view of the possible complications of the disease itself or from the dangers of inadequate treatment, I would feel that even a carefully selected group of cases of pernicious anemia would likely show a mortality of 150 per cent to 200 per cent. There are, of course, no figures available and this is strictly my personal opinion.

*SPRUE*

The various forms of sprue are all subject to the development of anemias. Usually the anemia tends to be of the hyperchromic macrocytic type. The results of treatment have been very poor and up to this time I would consider that all types of sprue would carry such a very heavy extra mortality that their consideration would be very doubtful indeed. The use of cortisone may have a decided effect in this disease from a clinical point of view. Whether or not the use of this drug can alter our attitude to some of the milder cases time alone will tell.

*ANEMIAS OF PREGNANCY*

The vast majority of these are of the iron deficiency type. Occasionally a macrocytic anemia is found which responds well to liver extract. After delivery, these patients appear to be normal. During subsequent pregnancies the onset of the anemia can be prevented by the use of liver extract throughout the term. It would not appear that these anemias of pregnancy have any bearing on longevity.

*HYPOCHROMIC ANEMIA*

The great problem in this group is the matter of accurately determining the underlying cause of the anemia. One's action in regard to insuring a person who has suffered from a hypochromic anemia would depend on the completeness of the investigation, the severity and duration of the anemia, the speed and the apparent permanence of the cure.

One must be very watchful in regard to the possibility of an ulcer or a varix or a carcinoma of the gastrointestinal tract causing chronic blood loss without visible hemorrhage. It may be well to call your attention especially to cancer of the ascending colon which can produce a marked anemia and very little else in the way of symptoms. Temporary recovery from the anemia caused by the growth has been noted even before surgical intervention. Other causes of chronic blood loss should be more apparent and therefore are less likely to be overlooked by the attending physician.

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: 49

Certain patients suffer from a severe recurrent idiopathic iron deficiency anemia. While these cases are made enormously better by adequate doses of iron, one must remember that there is a great tendency to relapse. We would be forced to consider whether or not the particular patient is intelligent enough to maintain adequate treatment. Considerable individual discretion must be used in assessing these cases.

There is a large group of truly secondary anemias due to conditions such as nephritis, other blood dyscrasias, deficiencies of various sorts, and chronic infections. It is impossible to generalize about these because of the varied etiology of the anemia.

Finally, there is that great conglomeration of mild anemias of unknown origin. These patients are usually female and have been known to have anemia for several years. Their hemoglobin estimation is in the neighborhood of 70 per cent. These patients may be mildly fatigued but are usually able to carry on light to moderate activities without much trouble. Treatment seems to have very little effect. If time and investigation have ruled out any serious disease, these patients do not appear to suffer from an appreciably increased mortality. One would tend to be guided, however, by the degree of anemia and the symptoms complained of by the patient.

### *BANTI'S DISEASE*

This condition can start at all ages but generally before 35. It now seems to be the consensus of opinion that splenectomy is of very little value. These patients ultimately die of cirrhosis of the liver or from hemorrhage. Many of them live and are comfortable for years, but potentially this is a very bad group indeed. I think that it would be imperative to wait for at least five years after operation in an early case of Banti's disease before one could consider it. The extra mortality would always be high.

### *HEMOLYTIC ANEMIA*

This disease is usually familial and is associated as a rule with an anemia. Deaths from this condition may occur

during an acute exacerbation of the disease. In any event, these patients are not well and in the absence of further treatment would likely show a high mortality. Splenectomy seems to cure a large proportion of the cases. If with this treatment the anemia and the jaundice disappear, they would probably be acceptable but hardly without allowing for an increased death rate.

Erythroblastosis fetalis carries a heavy immediate mortality for newborn children affected by this condition. If the patients are given small transfusions with Rh-negative blood about 65 per cent will recover. Recovery is complete and permanent.

Sickle cell anemia is apparently related to this condition. Most of these patients die by the third decade.

Elliptocytosis is a familial disease having 40 per cent of the red blood cells elliptical in shape. Provided the patients show no signs of hemolytic anemia, they appear to be normal.

There are several other hemolytic conditions, but the only one that I shall mention is hemoglobinuria. Those cases due to drugs such as sulphonamides or other toxic conditions such as burns will probably remain in good health. March hemoglobinuria seen in soldiers gradually disappears and seems to leave the patient in a normal state. Paroxysmal hemoglobinuria is brought out by exposure to cold and apparently is due to syphilis. The treatment of the syphilis seems to eradicate the condition. Paroxysmal, nocturnal hemoglobinuria is of unknown origin. It frequently leads to debilitating conditions such as venous thrombosis in one place and another. Anemia frequently is associated with the condition and the treatment is highly unsatisfactory. Prognosis I should think would be poor.

#### *APLASTIC ANEMIA*

These anemias are all fatal except when they are due to some form of drug therapy such as gold, arsenic, sulfonamides, radiation, etc. When aplastic anemia is said to be due to one of these drugs, it would be wise to make sure

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: 51

that the diagnosis is correct by waiting for one full year after recovery.

### *HEMOPHILIA*

While those who survive to be over 20 years of age may carry on for some years, this group seems to be very poor. I would consider that a proven case of hemophilia is subject to a prohibitively high mortality rate.

### *PURPURA HEMORRHAGICA*

In patients who have not had their spleens removed, about 25 per cent will remain in good health after one acute episode of purpura. This means that the remaining 75 per cent will have varying experiences in regard to relapses of this condition. Death from the disease is common among the group. Following splenectomy about 75 per cent seem to recover almost completely. One would feel like waiting about two years after operation before making any sort of attempt to gauge the mortality. It is probably high at best.

Secondary purpura will vary of course with the cause of the disease. The same remark applies to purpura from capillary damage. Secondary purpuras due to typhoid fever and such transient infections will be cured, whereas those from such diseases as leukemia will of course die. Capillary damage may result from many infections, from drug sensitivity, from vitamin deficiency, jaundice, etc.

### *HEMORRHAGIC DISEASE OF THE NEWBORN*

Hemorrhagic disease of the newborn shows itself between the fourth and tenth days of life. It is due to a low prothrombin content of the blood. As a result of this, hemorrhages may occur anywhere in the body but especially in the intestinal tract, the skin, and the central nervous system. About 50 per cent of the affected infants die of this condition. The use of vitamin K or repeated small blood transfusions will correct the condition in 50 per cent of the cases. Recovery is then complete except insofar as permanent damage may be left as a result of intracranial hemorrhage. Because of the possibility of this last complication, reports from at-

tending physicians would be necessary. Uncomplicated cases should show a normal mortality rate.

#### *INFECTIOUS MONONUCLEOSIS*

These patients will have a normal mortality after recovery, provided we feel that the diagnosis is accurate. The only possible error is in making an incorrect diagnosis in a case of acute leukemia.

#### *AGRANULOCYTOSIS*

These patients are apparently normal after a complete recovery. There is, however, the possibility of the patient's receiving a similar drug again or becoming sensitive in a similar manner to a different drug and having a very severe or fatal second attack.

#### *POLYCYTHEMIA RUBRA VERA*

This disease is eventually fatal although the patient may survive in a moderate state of health for many years. The length of life from the onset of the disease is between five and fifteen years.

#### *LEUKEMIA*

Leukemias of all types at the present time must be considered to be uniformly fatal in a short time.

#### *HODGKIN'S DISEASE*

Patients with Hodgkin's disease eventually die of their condition. Unfortunately the pathologist cannot always form an accurate diagnosis in cases of chronic gland enlargement. Mistakes of diagnosis may be made in both directions. The passage of a few years is not always a corrective feature, as cases of Hodgkin's disease with accurate diagnosis have been known to have periods of good health for up to ten or even twenty years. One must be very wary indeed of patients who have had enlarged lymph glands, without any evidence of acute inflammatory reaction, which have lasted for more than a few days or weeks and have responded to x-ray treatment. On the other hand, the glands of Hodgkin's disease will not disappear in two or three weeks without any treat-

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: 53

ment of any type. If the pathologist was at all suspicious of the case, I would act as though the diagnosis had been confirmed and the mortality rate thereby established.

There are, of course, other rare diseases of the blood and blood-forming organs about which it is very difficult to obtain any authoritative information and which because of their rarity do not particularly interest us today.

### REFERENCES

1. Metropolitan Life Statistical Bulletin 17:9 (Jan.) 1936.
2. Cameron, Townsend and Mills — Can. Med. Assoc. J. Vol. 65:241.

PRESIDENT YLVISAKER — Thank you, Dr. Falconer.

Dr. Jonathan C. Sinclair of the Canada Life Assurance Company will discuss Dr. Falconer's paper. Dr. Sinclair is one of our newer members and we are glad to welcome him in his first appearance on one of our programs.

DR. JONATHAN C. SINCLAIR — Doctor Falconer has reviewed in a very comprehensive fashion a field in which there have been many developments in recent years. In opening the discussion, I would like to comment briefly on three of the conditions described.

### *Hemolytic Anemias*

In our consideration of hemolytic anemias in adults, it is worthwhile, I feel, to subdivide them into (a) the congenital type and (b) the acquired type. In the former group, i.e., the congenital, there is, as a rule, a family history of the condition which gives rise to symptoms usually in adolescence or early adult life. At this time, investigation will reveal abnormal pigment metabolism as well as the characteristic spherocytosis and increased fragility of the red blood cells. The Coombs test is negative. It is worth bearing in mind that pigment stones may form in this disease and may occasionally give rise to biliary colic. With splenectomy, the increased breakdown ceases and the individual makes a dramatic recovery. From an underwriting point of view, if a case falls clearly into this group, I feel that two years after

successful removal of the spleen such a case will usually be standard. The acquired type may be differentiated from the congenital form by the absence of family history, the inconstant demonstration of abnormal fragility and spherocytosis of the red cells and the finding of an antibody adherent to the red cells, i.e., a positive Coombs test. Acquired hemolytic anemia is often an expression of some other disease which may be overshadowed by the hemolytic process. Therapy with ACTH or cortisone has given rise to remissions but it is too soon to properly assess the results. Splenectomy has also been tried in certain carefully selected cases. However, there is a distinct tendency to relapse, and for this reason I feel that this group is, as a rule, uninsurable.

#### *Purpura Hemorrhagica*

Our problem with reference to purpura hemorrhagica is becoming more complex since many of these patients are now being treated with ACTH or cortisone in preference to splenectomy. The response to this form of therapy appears encouraging. A period of observation for some years will be necessary before the results can be applied to underwriting.

#### *Pernicious Anemia*

Several investigators during the past decade have commented upon the increased incidence of gastric carcinoma in patients with pernicious anemia. Kaplan and Rigler (1) reported an incidence of 12.3 per cent when they reviewed a series of autopsies on 293 patients with pernicious anemia. This they noted to be three times as great as the incidence of gastric carcinoma in a large autopsy study of a comparative age group.

Mosbech and Videbaek (2) last year reported a follow-up study of 219 women and 82 men under treatment for pernicious anemia in Copenhagen during the period 1928 to 1949. After following 220 patients for more than ten years, they conclude that the chances of survival for the female patients are the same as for normal women. With male patients, there appears to be a slightly higher mortality. In recording the cause of death in 115 cases of pernicious anemia, they noted

## BLOOD DYSCRASIAS: CURRENT CONCEPTS: 55

that 21 died from carcinoma with gastric carcinoma responsible in 14 cases (8 female and 6 male). The expected number of deaths from gastric carcinoma in a corresponding normal group is 5. In this series, there would certainly appear to be an increased incidence of gastric carcinoma. Deaths occurred in 83 women and 32 men, a total of 115. The expected number of deaths for a corresponding group of normal persons is 117 (89 female and 28 male). Thus, despite the number of cases of gastric carcinoma recorded, the overall mortality for these patients corresponds very closely with that of a normal group of the same age and sex distribution.

I fully agree with Dr. Falconer that, in underwriting such a condition, we should be certain

- 1 That the diagnosis is accurate
- 2 That neurological involvement, should it be present, is mild in degree
- 3 That adequate therapy is being given
- 4 That the individual is well adjusted and cooperative in maintaining treatment

With such careful selection, extra mortality would be moderate.

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2. Mosbech, J. and Videbaek, A.: B. M. J., 2:390, 1950.

**PRESIDENT YLVISAKER**—I want to thank Dr. McCue, Dr. Falconer, and Dr. Sinclair for their splendid presentations. They have all been very cooperative and helpful.

Our afternoon program is devoted altogether to our cardiovascular problems, because we meet with these problems so frequently in our selection work. Among our policyholders, cardiovascular conditions cause between 65 and 70 per cent of all deaths and over 50 per cent of all total disability. Among our applicants for insurance, these same conditions cause over

60 per cent of all our rejections and about 50 per cent of our substandard ratings.

We are fortunate that our Life Insurance Medical Research Fund is using all its resources for the study of our cardiovascular problems. We have with us today the Executive Director of this Fund. We all know Dr. Francis R. Dieuaide and how well he is directing this work. I am glad to call on him to discuss "Current Progress in Cardiovascular Research."

DR. FRANCIS R. DIEUAIDE — Dr. Ylvisaker, Members of the Association: I should make it clear that in the brief summary which I have prepared to give you this afternoon I have not adhered solely to actual pieces of research that the Life Insurance Medical Research Fund has been directly concerned with, because I thought it would be more interesting to follow the title which your chairman gave me, "Current Progress in Cardiovascular Research," and later to say a little about the Medical Research Fund as such.

## CURRENT PROGRESS IN CARDIOVASCULAR RESEARCH

FRANCIS R. DIEUAIDE, M. D.

*Scientific Director, Life Insurance Medical Research Fund*

The vigorous attack now being made by research workers on cardiovascular disease is most encouraging. The prospect for the solution of some of the problems involved in the prevention of hypertension, arteriosclerosis, and rheumatic fever is far better today than it was five years ago. It is noteworthy that the development of an effective, practical means of preventing any one of these diseases will be as great an advance as has ever been made in public health.

On this occasion, only a few of the many recent developments in the cardiovascular field can be mentioned. Those that seem most stimulating in relation to the main types of disease have been selected.

Evidence has accumulated indicating that a disturbance in lipid metabolism plays a basic role in arteriosclerosis (1). A great stimulus to work in this field has been provided by the discovery of Gofman and his collaborators (2) that an abnormal concentration of certain lipoproteins carrying cholesterol occurs in the blood of persons who have suffered myocardial infarction (the  $S_f$  12-20 class, as measured in the ultracentrifuge). Though doubts exist as to the interpretation and especially the prognostic applicability of this information, it is an important contribution to the growing knowledge of lipid disturbances in arteriosclerosis.

It is necessary, however, to learn a great deal about the normal metabolism of cholesterol and fats, since knowledge of this subject has been fragmentary. Essential knowledge is accumulating of the synthesis, turn-over, and degradation of these substances in the body (3). It is now known that cholesterol can be built up, not only in the liver, but also in various other tissues, including the arteries themselves

(4). The basic, if not the only, material used is acetate which is provided by both fats and proteins in general. Some four or five times as much cholesterol is turned over in the body as is consumed in the diet.

Further needed knowledge concerns the transportation and particularly the deposition of cholesterol and fats. Dr. Gofman's finding may be expected to lead to early increases in our knowledge of lipid transportation in the body. Active research is already in progress on the factors involved in the local deposition of these substances, which is an outstanding characteristic of arteriosclerosis. In connection with its pathogenesis, this is an important matter, because arteriosclerosis is a focal disease rather than a general change. Local factors must play some role, so they are being studied with care.

As to the mechanisms involved in setting up lipid disturbances, we are still much in the dark. There are observations which suggest that various hormonal malfunctions may play a role, but it does not seem that any of the well known endocrinopathies, such as hypothyroidism, can be responsible for arteriosclerosis. Available evidence does not substantiate the idea that age alone is the cause of the disease. Although there is a general increase in susceptibility up to middle age, susceptibility apparently does not increase in old age.

A large volume of work is being done on the role of dietary cholesterol (5), but it cannot yet be said how important this factor is. From the practical point of view, we can agree that individual physicians may properly prescribe diets low in fat and cholesterol for selected patients on an experimental basis. There is, however, no satisfactory basis for any general program of dietary reform in this direction.

Emphasis has often been laid on the point that hypertension may have many different causes. For some years, however, as a result of the work of Goldblatt, interest has been largely centered in the kidneys as the seat of the principal disturbance underlying hypertension, at least in many cases. It is of

## PROGRESS IN CARDIOVASCULAR RESEARCH 59

special interest that two recent lines of work appear to be coming together, with the probable result that a new and subordinate role will be assigned to the kidneys.

The work of Grollman (6) and others has established the fact that hypertension can be produced in animals completely deprived of their kidneys. In these instances, hypertension cannot be due to the production by the kidneys of a vasoconstrictor substance. By a completely independent approach, Handler and Bernheim (7) have had results strongly suggestive that in so-called renal hypertension the kidneys are failing to excrete a hypertensive agent which is normally removed from the body as fast as it appears. This work holds out the fascinating possibility that a new urinary test of far-reaching significance may be added to clinical pathology.

The results of Handler and Bernheim also show that "renal" hypertension is dependent upon an adequate protein intake and that such hypertension cannot occur on a diet deficient in protein because of inadequate production or release of ACTH by the pituitary gland. Since ACTH exerts its effect on the adrenal cortex, we have thus been led back to the adrenal glands which first entered the picture many years ago through the effects of the adrenal medullary secretion, epinephrine. There, at the moment, this matter rests pending further investigation.

As to treatment, it has been shown beyond question that prolonged severe restriction of dietary protein and salt produces improvement in many patients with hypertension (8). Up to a certain point the work of Handler and Bernheim furnishes an explanation of the action of protein restriction. The majority of observers are agreed, however, that protein and salt restriction as applied in the rice diet so far are much too troublesome for widespread use. Experiments are in progress which may result in a regimen free from the objections inherent in the rice diet as hitherto employed.

Recent work has added little to our knowledge of the value of sympathectomy in hypertensive disease. It is generally agreed that a small group of patients are much benefited by

the various operative procedures, but also that it is extraordinarily difficult to foretell what patients will be benefited. A large volume of research has been done on drugs which block sympathetic nerve action at various levels (9). As yet, none of these agents has been found very helpful in the diagnosis or treatment of hypertension. The hypotensive effects of certain drugs related to veratrum have been found sufficiently encouraging to warrant extensive study (10).

Advances in knowledge bearing on the mechanism of rheumatic fever consist mainly of precise information about substances produced by the streptococcus during its growth (11). Some of these products might be responsible for rheumatic fever by acting as antigens. Bodily reactions to their presence are being studied (12). We are still in the dark as to why a few persons develop this disease out of the great number who suffer streptococcal infections. It is of special interest to note that new evidence has been obtained which indicates that the incidence of rheumatic fever is declining (13).

The value of using ACTH or cortisone in rheumatic fever is still a subject on which opinions differ. Symptoms are suppressed, but the evidence so far does not favor the conclusion that heart disease is prevented or cured (14).

An important recent development is the demonstration by investigators working for the Armed Forces Epidemiological Board (15) and by others that treatment of streptococcal respiratory infection with penicillin prevents the subsequent occurrence of rheumatic fever.

After a lapse of some twenty years, further work has been done on the problem of surgical treatment of mitral stenosis (16). Enough success has been achieved to be encouraging, particularly in the light of the possibility mentioned below that current research may provide a practical mechanism for excluding the heart from the circulation while the surgeon operates.

In connection with general diagnosis and therapy of cardiovascular disease, one should note developments in vectorcardiography, electrokymography, and ballistocardiography.

## PROGRESS IN CARDIOVASCULAR RESEARCH 61

A chemical assay of digitalis has been developed, which gives results parallel to the potency of the drug in man (17). Under the leadership of Gibbon in Philadelphia (18), a mechanical heart-lung apparatus has been developed which offers promise of making it possible for the surgeon to perform operations on the heart in a bloodless field.

Six years ago, life insurance companies provided the first substantial source of funds devoted to the support of cardiovascular research by organizing the Life Insurance Medical Research Fund. Other agencies, notably the American Heart Association and the National Heart Institute, have since joined in making available more nearly adequate support for research in this vital field.

The Annual Report of the Life Insurance Medical Research Fund for 1950-51, now in press, shows that during the past year the Fund supported 85 research programs and 33 research fellowships. Contributions to the fund for 1951 amounted to \$776,075. Since its establishment in December 1945, the Fund has allocated \$3,931,421 to the support of cardiovascular research. Grants and fellowships have aided research in 85 institutions located in 33 states, 4 Canadian provinces, and 4 other countries. The new Annual Report provides full information about the results of research aided by the Fund.

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PRESIDENT YLVISAKER—Thank you very much, Dr. Dieuaide. We are all glad to hear of the splendid progress which is being made by the Life Insurance Medical Research Fund under your direction.

I am particularly glad that our next speaker could hear your remarks, Dr. Dieuaide. Some of you will know that Dr. Marvin spent much time in the early forties trying to interest life insurance companies in cardiovascular research and in the program of the American Heart Association. Your remarks will help him to realize that his work has borne fruit and will stimulate all of us to support this research work even more in the future than we have in the past.

Dr. Marvin was President of the American Heart Association in 1949 and 1950. At the meeting of this organization in June, he was one of the three physicians who received the Gold Heart Award, which is one of the most significant distinctions in cardiovascular medicine.

I should like to read the citation given to Dr. Marvin. "The Gold Heart is but a token of our recognition of

## PROGRESS IN CARDIOVASCULAR RESEARCH 63

Dr. Marvin's devoted and unselfish contributions to the growth and development of the American Heart Association over the past quarter of a century. In fact, the life of the Association has been closely interwoven with his. If anyone deserves the name of Mr. American Heart, that person is Jack Marvin. As president of the Association in 1949 to 1950, he climaxed a career of service in many important organizational posts. He served as executive secretary of the Association from the early thirties until 1946. He had the responsibility—and leadership more than anything else—of planning and executing the conversion of the American Heart Association from a purely scientific medical organization to its present position, a voluntary organization devoted to research, education, and community service in the cardiovascular field.

"Dr. Marvin has contributed greatly to our scientific knowledge with over forty articles. He has made a most valuable contribution to the popular literature in a lay educational program in the cardiovascular field as co-author and editor of the book, 'You and Your Heart,' published last year."

Those of you who have heard Dr. Marvin also know that he is a wonderful teacher. I have heard him on several occasions in the past, and his talks are just as alive to me today as they were at the time he first gave them. When we discussed the subject that he was to present today, he suggested the topic that is on our program, "Differential Diagnosis of Chest Pain." I think that in doing so he selected a topic that is of very great importance to us. It is disturbing for us in our work to have so many applicants come to us with a history of chest pain. Naturally, if we cannot exclude the serious possibilities of that pain, we are forced to decline the individual for insurance.

Dr. H. M. Marvin will talk to us on "The Significance of Chest Pain." A history of chest pain gives us one of our most difficult underwriting problems and I am sure Dr. Marvin will help us with his discussion of this problem. Dr. Marvin.

## THE DIFFERENTIAL DIAGNOSIS OF CHEST PAIN

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It is certainly evident to all of you that it would be impossible in the time allotted to discuss adequately or even sketchily all of the many conditions that may cause pain in some part of the chest. Many of these are quite rare, some of them are relatively unimportant, some can be recognized instantly, and therefore need not be considered in the differential diagnosis (for example, herpes zoster). Some are of little or no significance in terms of actual disease, such as the pain that may follow mild trauma to the wall of the chest. With your permission, therefore, I shall restrict my discussion to pain in the anterior aspect of the chest and will discuss chiefly those causes that seem to me of major importance. No apology is necessary for beginning with pain arising in the heart, inasmuch as this is surely the most important, not only for the reasons emphasized by Dr. Ylvisaker a moment ago, but also because of its frequency and its potential or actual seriousness. With few exceptions, cardiac pain is the doctor's first suspicion when confronted by an example of severe pain in the anterior chest, and his first diagnostic studies are usually directed toward proving or disproving that suspicion.

As I think all of you know, it is customary today to divide pain arising from inadequacies of the coronary blood flow into three main groups, namely, angina pectoris, coronary thrombosis with cardiac infarction, and coronary insufficiency. There is another subgroup currently known as subendocardial necrosis, which will not be discussed.

It is recognized clearly that the terms myocardial infarction and coronary thrombosis are not synonymous. One may occur without the other. But with your permission, the terms coronary thrombosis, coronary closure, cardiac infarction,

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 65

and myocardial infarction will be used synonymously throughout my talk this afternoon.

The first of these three has many names, the commonest being angina, angina pectoris, anginal pain, angina of effort, anginal heart failure, cardiac pain and coronary pain. In most instances it has a single manifestation, namely, discomfort or actual pain in the anterior chest, sometimes radiating widely from its initial location. Through generations of medical experience and thousands of vivid descriptions, it has become established that there are certain characteristics which are so nearly constant and invariable that they permit a confident recognition of the condition. I think the most important characteristics are these: first, it is almost always a sense of constriction, tightness or pressure rather than actual pain, although it may rise to the level of severe pain if the provocative factors continue.

Second, it is located beneath the sternum, although commonly described by doctors as precordial. If the term precordial is used to designate that portion of the chest overlying the heart to the left of the sternum, then it is quite inaccurate and misleading to describe anginal pain as precordial. It is accurate and very important to describe it as substernal.

Third, if it radiates from the point of its initial and major location, it usually goes to the throat, the side of the neck, the shoulder, and down the inner aspect of the upper arm as far as the elbow; sometimes down the ulnar aspect of the forearm and into the little and ring fingers. Other areas that may be involved include the angle of the jaw, the teeth, sometimes the interscapular area. Radiation to the left is far more frequent than to the right.

Fourth, it is brief in duration, seldom lasting more than two or three minutes when caused by physical effort, and sometimes subsiding within fifteen to thirty seconds. It is not at all uncommon for patients to speak of their pain as lasting ten or twelve minutes. The great majority of them, if asked to time it accurately with a watch, will later report that it seemed like ten or fifteen minutes, but was actually only two minutes.

Fifth, it is closely and almost invariably related to increased work of the heart, the commonest provocative factors being physical exertion and emotional excitement.

Sixth, it is reproducible. The same kind and intensity of discomfort will follow repetition of the same effort again and again, and the discomfort will disappear each time in the same number of minutes or seconds following the termination of exercise.

Seventh, it may be present even in severe form in the complete absence of any abnormal findings in the physical examination, the usual routine laboratory tests, x-ray study of the heart, and the resting electrocardiogram.

Eighth, it is usually relieved very promptly by the administration of nitroglycerin, although sometimes not more quickly than by rest alone. It is far more important to emphasize that it is usually prevented completely if nitroglycerin is given in advance of physical effort or the exposure to emotional excitement.

Of these eight characteristic features, several should receive particular emphasis because of their importance in distinguishing this condition from others. I would ask you to keep especially in mind the substernal location, the brief duration, the radiation in to the *inner* aspect of the arm, the reproductibility by repetition of the same effort, the relationship to increased work of the heart, and the preventive action of nitroglycerin.

I wish there were time to review for you the fascinating story of the discovery of the cause of this extraordinary symptom, but must content myself with the bare statement that there appears to be unanimous agreement among competent authorities that the cause is to be found in a discrepancy between the needs of the heart for blood and oxygen, on the one hand, and the available supply on the other. In patients with angina, discomfort may be expected to arise whenever the demands of the heart for blood are greater than the supply available at that moment. Inasmuch as there

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 67

are two factors involved—supply and demand—it is clear that a discrepancy between them can arise whenever there is a change in one without a corresponding and simultaneous change in the other. It is not surprising, therefore, that we find anginal pain occurring in those conditions which increase the work of the heart without a simultaneous increase in its blood supply; such conditions as hyperthyroidism, paroxysmal tachycardia, physical exercise, emotional excitement (which is the equivalent of exercise), exposure to a cold atmosphere and, in certain instances, aortic insufficiency.

It is equally clear that a discrepancy will arise if the supply of blood and oxygen to the heart is diminished without a corresponding and simultaneous decrease in the work of the heart. Thus we find, for example, that typical and unmistakable substernal squeezing pain on effort will occur in young and otherwise healthy individuals if there is moderately severe anemia. The substernal pain on effort promptly disappears when the blood count and hemoglobin have been restored to normal levels. There is considerable divergence of medical opinion as to the occurrence, frequency, and importance of spasm of the coronary arteries or arterioles, but if this does in fact occur in human beings under physiologic conditions it would obviously cause a decrease in the coronary blood flow. Apparently such spasm could explain many of the reported cases of "tobacco angina", and it is tempting at times to assume reflex spasm of these vessels to explain attacks of pain that otherwise remain obscure. But in the great majority of cases it is unnecessary to invoke this explanation. By far the commonest cause of anginal pain is a reduction of the blood flow due to narrowing of the coronary arteries by those changes in the intima known as atherosclerosis.

Between simple angina, which has just been described, and coronary thrombosis with cardiac infarction, there are striking differences which usually permit a confident diagnosis of one or the other. Not only does coronary thrombosis give rise to much more severe pain of longer duration, but also there are subsequent findings which usually serve to

identify it without difficulty. I refer to such things as the pallor, weakness and sweating which commonly accompany the onset of the pain, but particularly to the later appearance of fever, leukocytosis, increased sedimentation rate and, most important of all in many instances, the highly distinctive changes which take place in serial electrocardiograms. However, it is well to emphasize that the relatively abrupt onset of anginal pain should always arouse the suspicion that cardiac infarction might have occurred several days or a week before. Not infrequently one encounters patients, some of them highly intelligent and observant individuals, whose only symptom is mild or moderate substernal squeezing on walking. Yet electrocardiograms on these patients sometimes show unmistakably that cardiac infarction has occurred in the very recent past.

There is a third condition that may be regarded as midway between angina and coronary thrombosis. It is characterized in many instances by pain which is longer in duration than that of angina and shorter than that of coronary thrombosis. It may or may not be related to effort. It may or may not be relieved by nitroglycerin. The changes indicating tissue destruction just mentioned, namely, fever, leukocytosis, increased sedimentation rate, usually do not occur, or if they do occur they are borderline in degree. Changes in electrocardiograms characteristic of cardiac infarction do not occur. If changes are present, they usually consist of traction depression of the ST segments. It is not my intention to discuss electrocardiography in any detail, because I realize that this is to be done competently and interestingly by the speakers who follow.

This condition, as you all know, is commonly designated as coronary insufficiency or coronary failure. These terms are far from ideal. They are not precise, for the discomfort which they designate covers a wide range of duration and intensity, but I think their introduction has served a useful purpose because it has emphasized the impossibility of regarding all cardiac pain as representing either simple angina or coronary thrombosis with cardiac infarction.

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 69

The diagnosis of coronary insufficiency is sometimes made on the basis of the history and the resting electrocardiograms taken shortly after the onset of symptoms. It is made far more frequently, as you know, on the basis of electrocardiograms taken under conditions which subject the heart to stress of one form or another. There are three such tests in frequent use today. One was designed by Dr. Arthur Master of this city, and is commonly known by his name. This consists of having the patient take a designated number of steps across a standard two-step platform, the number being determined from tables based on the patient's age and sex, height and weight. Electrocardiograms are taken at the conclusion of the effort, and the test is said to be positive—and coronary insufficiency is said to be present—if the electrocardiogram shows certain changes, particularly depression of the ST segments in the limb and precordial leads.

A second test is that devised by Dr. Robert Levy of this city, and known as the anoxemia test. It consists of having the patient breathe 10 per cent oxygen for a period of twenty minutes; electrocardiograms are taken subsequently.

The third test, which I personally prefer at this moment, consists of having the patient walk over the two-step platform, not for any specified number of trips based on the factors just named, but rather to have him walk until he develops uncomfortable breathlessness or pain—or in the absence of these symptoms, to make fifty complete trips. Those who devised this test have the patient hold an ice cube in each hand in order to make the test a little more severe by elevation of the blood pressure. One important feature of this test, it seems to me, is its emphasis on taking electrocardiograms not immediately after the conclusion of effort, but several minutes later. In fact, it is recommended that electrocardiograms not be taken until the heart rate has returned to its pre-exercise level. It has been my custom to take electrocardiograms every two minutes after the conclusion of effort. Sometimes the electrocardiogram taken

immediately upon the conclusion of effort shows no significant changes, whereas one taken three, four or five minutes later may show very striking changes.

These three forms of cardiac pain, then, arising from inadequacies of the coronary blood flow, may serve as our starting point, as the fundamental pattern with which to compare other kinds of pain.

Next, I should like to speak briefly of the pain of two different kinds of pericarditis. Ordinarily, the pain arising from acute fibrinous pericarditis is not confused for very long with either angina or cardiac infarction, because the pain usually differs in its character and location from that of coronary inadequacy. As you know, this type of pericarditis usually occurs as a result of rheumatic fever, tuberculosis, or infections of the pleura, lungs, or mediastinum. Not infrequently, the circumstances will indicate the exact diagnosis, for example, when a pericardial friction rub appears in a child who has obvious rheumatic fever. If not, the other characteristics will usually differentiate it.

The pain is ordinarily sharp rather than dull. It is almost always aggravated by deep inspiration, by coughing or by movements of the body. It is sometimes beneath the sternum, but far more frequently is more diffusely located over the whole precordial area. It may vary in location from day to day, or even from hour to hour. If a friction rub is heard in a youthful patient, the diagnosis becomes quite certain. If it is heard in an older person, for example, a man in his fifties, it is of no differential value because, as you know, cardiac infarction in people of that age not infrequently is associated with a friction rub during the first several days.

Electrocardiograms may be of very great importance in the diagnosis, particularly if they are obtained early in the course of the disease, because they will usually display the pattern that is characteristic of this condition, namely, elevation and upward concavity of the ST segments; secondly, a failure of the sign that is characteristic of cardiac infarction, namely, reciprocal or opposite changes in leads 1 and 3. Most

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 71

of you are aware that in cases of anterior cardiac infarction there is elevation of the ST segments in leads 1 and 2, and reciprocal depression of this segment in lead 3. In pericarditis, the same kind of change takes place in all three leads.

However, when we come to the other type of pericarditis, the so-called acute, benign, nonspecific pericarditis, it must be confessed that there is sometimes great difficulty in differentiating this from cardiac infarction. This disease, as you may know, tends to occur chiefly among the younger age groups, which often helps in the differential diagnosis. It is a relatively benign disease in most people, as the name indicates. The clinical course is usually a mild one. In perhaps the majority of cases, there is no difficulty whatever in making the diagnosis. That is particularly true if electrocardiograms are taken shortly after the onset of symptoms; but, unfortunately, sometimes electrocardiograms are not taken for a week or two after the initial pain, in which case there may be difficulty.

It so happens that practically all the patients I have seen with this condition have been between the ages of eighteen and thirty. That, in itself, makes one look with great suspicion upon a diagnosis of cardiac infarction because although it does occur in people of that age group, particularly in men, it is still a rare disease under the age of thirty.

If the electrocardiograms are taken early, they will usually show the classical pattern of pericarditis, just described briefly, namely, elevation and upward concavity of the ST segments in the limb leads, and the absence of a reciprocal relation between leads 1 and 3. But if they are not taken for a week or ten days, one then finds electrocardiograms showing deep inversion of the T waves exactly like that which one finds in cardiac infarction, with the exception just mentioned—that there is an absence of the reciprocal relationship which almost always obtains in cardiac infarction. Perhaps I can express it otherwise and more simply by

saying that the fully developed electrocardiographic changes in a moderately severe case of acute, benign, nonspecific pericarditis will be exactly like the electrocardiographic changes of a patient who has had infarction of the anterior, posterior, and lateral walls of the left ventricle. But the relative youthfulness of most of these patients, and the rather benign course of the disease are simply not consistent with the belief that such extensive cardiac infarction has occurred.

It would be very foolish of me to say or to imply that the differentiation between these two conditions is always easy, for it is not; but I think it can usually be made with confidence if the possibility of pericarditis is kept in mind.

Dissecting aneurysm of the aorta not infrequently gives rise to symptoms and a clinical picture that resemble cardiac infarction very closely indeed. In fact, if the patient dies within a few hours after the onset of symptoms, it may be absolutely impossible to determine without postmortem examination which of the two conditions was present. If the patient survives, there may be features which point clearly toward progressive separation of the aortic coats rather than toward cardiac infarction. This condition commonly occurs in hypertensive individuals, and the blood pressure usually remains high throughout the early hours or days after the onset of symptoms, whereas in cardiac infarction of comparable severity one of the constant features is a dramatic and considerable fall in blood pressure.

The pain of dissecting aneurysm is, in my experience, probably the most agonizing to which human beings are subject. It often requires heroic doses of morphine, and even these sometimes give little or no relief. That, in itself, may arouse suspicion as to the correct diagnosis.

If the dissection begins in the ascending aorta, or in the arch, and progresses backward toward the heart, it may eventually result in closure of the mouth of one of the coronary arteries, and this will give rise to an electrocardiographic pattern of cardiac infarction. Otherwise, the electrocardiograms do not show the typical pattern of infarction, and this

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 73

may be an important diagnostic point. The dissection may proceed backward so far as to result in separation of the aortic valve leaflets, and this will give rise to a diastolic murmur which was not present earlier.

If the dissection proceeds in the other direction, that is, through the arch and into the descending aorta, sometimes there is a dramatic change in the quality and location of the pain from hour to hour and from day to day. This in itself, should stamp the condition unmistakably as dissecting aneurysm and not coronary thrombosis; because such changes in the location and character of the pain, so far as I know, practically never occur with cardiac infarction.

Finally, the pain of dissecting aneurysm usually lasts very much longer than that of cardiac infarction, sometimes for many days, regardless of the use of opiates.

Pulmonary embolism, similarly, may give rise to pain and to other clinical features which make one strongly suspect cardiac infarction. In both these conditions, there may be very severe substernal pain, cyanosis, dyspnea, tachycardia, and shock—symptoms similar in character and severity to those commonly associated with closure of a large coronary artery. In some instances the attendant circumstances give the clew to the diagnosis, and these should always receive due consideration. For example, if these symptoms arise in a woman who has recently given birth to a child, in a patient recently subjected to surgical operation, in a patient known to have had phlebitis or extensive varicose veins, in a patient with auricular fibrillation, or in any patient who has been long confined to bed, pulmonary embolism would be the tentative diagnosis.

On the other hand, if these symptoms occur in a man in his fifties, in a patient known to be hypertensive, or one known to have had angina, coronary thrombosis would be one's first thought. Electrocardiograms may be of crucial importance, for I think it is reasonable to say that if complete electrocardiograms are taken, that is, three standards and three unipolar

limb leads and five or six precordial leads they will reveal cardiac infarction, if it has occurred, in almost all cases.

There is no single pattern that is so distinctive of pulmonary embolism. Some eight or ten different patterns have been described in medical reports as occurring after pulmonary embolism; but the very multiplicity of such changes in these graphic curves means, I think, that the one important feature is the absence of those changes which would permit a confident diagnosis of cardiac infarction.

It is perhaps unnecessary to emphasize to this audience that the terms "pulmonary embolism" and "pulmonary infarction" are not synonymous, for embolism is not necessarily followed by infarction of the lung tissue. Pulmonary embolism is characterized by the symptoms just mentioned. If infarction follows it, the symptoms of infarction are usually those of acute pleurisy and are not often confusing as to diagnosis. The location and size of such infarcts can usually be demonstrated without difficulty in x-ray films unless they happen to be located directly behind the heart.

For the next topic and the comments that I wish to make upon it, I am indebted to one of my dear friends who is known personally to many of you, and who spoke before this Association about two years ago: Dr. Tinsley R. Harrison, now Professor of Medicine at the Medical College of Alabama in Birmingham. He has kindly sent me the manuscript of a paper which will appear in *Circulation*, the official *Journal* of the American Heart Association, within the next month or two months. The title of his paper is "Chest Pain in Association with Pulmonary Hypertension: Its Similarity to the Pain of Coronary Disease."

In order not to do injustice to Dr. Harrison's admirable discussion, I propose with his permission to read a few excerpts from his paper. I wish to commend it with all my heart to each of you and to all your examiners, because it emphasizes something that seems to me of the utmost importance from the standpoint of medical teaching, not only to those engaged in practice of internal medicine, but also to those who

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 75

specialize in the field of cardiovascular disease. I am quite sure that in past years I have often made the mistake which Dr. Harrison says in this paper he, too, has made. I shall now read excerpts from that paper which you will all have an opportunity to read for yourselves in full, including the very illustrative and helpful case histories, within the next month or two months.

"This communication deals with a type of chest pain which is not uncommon and which is likely to lead to an erroneous diagnosis of coronary arterial disease. In the patients we have observed, the one constant feature other than pain has been clinical evidence of increased pressure in the pulmonary vascular circuit. Hence, the term pulmonary hypertensive pain is employed, but with the reservation that more complete knowledge of the exact pathogenesis may make a different name desirable.

"Pulmonary hypertensive pain may resemble angina pectoris not only as regards location, radiation, intensity and quality of the discomfort, but also as regards the tendency to be initiated by physical exertion. In some patients the pain may be associated with outspoken electrocardiographic changes. Therefore, the diagnosis of myocardial infarction shown is likely to be made.

"The differentiation, while occasionally almost impossible, can usually be made with accuracy if one is familiar with the various clinical features of this syndrome. Most of the patients whom we have thought to be suffering from pain as a result of pulmonary hypertension have had one of the following: 1—lesions of the mitral valve; 2—certain congenital anomalies of the heart; 3—primary diffuse disorder of the lungs, especially asthma and emphysema; 4—disorders of the pulmonary artery, more particularly, embolism.

"Not all patients with these several disorders complain of pain. On the contrary, it is absent in the majority of instances. It is likewise rarely if ever encountered in patients with pulmonary hypertension secondary to left ventricular failure. Whether its presence or absence is dependent on the degree

of pulmonary hypertension or on other factors is uncertain at present. Important in diagnosis is the realization that any disease which is capable of causing pulmonary hypertension *may* produce pain, and to demand unequivocal evidence before concluding that such a patient has coronary disease.

"Certain features may serve as differential guides: 1—a history of long-standing cough; 2—cyanosis, either persistent or intermittent, and usually present during the attack of pain; 3—association of the pain with dyspnea; 4—the presence of pain on breathing; 5—clinical evidence of right ventricular hypertrophy (this is one of the most important of all signs); 6—evidence of tissue destruction, such as fever, leukocytosis and rapid sedimentation rate, occurs only when there is co-existent infarction or infection of the lungs; 7—nitroglycerin has little or no effect on pulmonary hypertensive pain, and striking effects on angina.

"We have seen a number of patients diagnosed fifteen or twenty years ago as anginal patients, but who obtained little or no relief from nitroglycerin. These patients we now recognize as having pulmonary hypertensive pain."

That concludes the quotation from Dr. Harrison's paper. He discusses at some length the probable mechanism of production of this pain, and comes to the conclusion that it is probably not arterial anoxia. It is not due to diminished coronary blood flow, to pulmonary arteriosclerosis or so-called right ventricular angina, but almost certainly is due to distention of the pulmonary artery; and in his opinion, the pain, therefore, is analogous to the pain known as migraine. In every autopsy in his series of cases, there was dilatation of the pulmonary artery.

There is another condition that leads not infrequently to a mistaken diagnosis of coronary thrombosis with cardiac infarction, especially during the first few hours after the onset of symptoms. I refer to spontaneous pneumothorax. It is recognized that in the great majority of instances this condition does not give rise to pain of the character or severity of that associated with cardiac infarction, but in some the pain

may be of agonizing intensity, requiring several large doses of morphine before relief is obtained. In addition, these patients often display striking pallor, sweating, great dyspnea, tachycardia, faint heart sounds, and fall in blood pressure. In other words, they display precisely the same clinical picture that one associates with cardiac infarction.

In all the patients I have encountered in whom the suspicion of cardiac infarction arose, the condition has been in relatively young men, far below the average age at which cardiac infarction ordinarily occurs. It is known that infarction can occur in young men, but it is still relatively rare.

Theoretically the diagnosis of spontaneous pneumothorax should be made with great ease, and within a few seconds, on the basis of physical examination. At the risk of bringing lasting discredit upon myself, I wish to go on record as saying that I think its recognition on the basis of physical examination is often extremely difficult. The changes that take place in the percussion note and breath sounds on the affected side are sometimes so slight as to escape detection. In self defense, let me say this. Many years ago, perhaps twenty-five, I encountered a young man about twenty-three years of age, in whom his physician had made a diagnosis of cardiac infarction. I saw him within two or three hours. While I did not think he had infarction, I did not know what he did have, because the clinical features were those just enumerated. On physical examination I failed to detect changes in the lungs which would lead me to the correct diagnosis, and did not make the diagnosis until I had fluoroscoped the patient. I then felt so ashamed and chagrined that I called in succession, two excellent internists who had offices in the same building, gave them the history, and asked them to examine the patient. Neither of them suspected pneumothorax after careful examination of the heart and lungs. I have had similar experiences in three other patients; and while, theoretically, the percussion note and the absence of breath sounds or their great diminution on the affected side should give the clew, I wish to say that in many instances in my experience, there has been practically no change be-

tween the side where the lung has collapsed and the side where the lung is still inflated. Of course, the diagnosis is very easy on the basis of fluoroscopic examinations or x-ray films. If, for any reason, these cannot be obtained immediately, electrocardiograms may be of negative value in that they do not reveal the characteristic changes that one would expect if cardiac infarction had occurred.

It has been stated very often that the pain in the chest associated with neurocirculatory asthenia sometimes resembles anginal pain so closely as to lead to great confusion in the diagnosis. It is my own belief that this confusion will occur relatively seldom if one keeps clearly in mind the various distinctive features of anginal pain which I mentioned at the beginning of my talk.

Neurocirculatory asthenia is today commonly regarded as a form of anxiety neurosis, and, the patients who are subject to it usually display clear evidences of emotional disturbances. If such a patient is observed while pain is present, it usually becomes clear that it lacks all or most of the characteristics of angina. It is seldom substernal in location. At least, it is seldom confined to the area beneath the sternum, as angina very commonly is. Indeed, in my own experience which, I must confess, does not include thousands of cases, I have never encountered a single instance of neurocirculatory asthenia in which the pain remained localized beneath the sternum, although the substernal area may have been involved in pain of wide distribution. It is usually much more diffuse over the left side of the chest, and not infrequently it has its maximal intensity around the apex of the heart, or even out in the anterior axillary line. It lasts for many minutes or many hours, which angina does not. Often there is residual tenderness over all or most of the affected area for hours after the acute discomfort has subsided. It often occurs in the absence of physical exercise or emotional excitement, although it may be induced by these factors and may continue for long periods after they have ceased to operate. Moreover, if one observes these patients, there are usually clear evidences of an anxiety state, such as wide dilatation of the

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 79

pupils, profuse sweating, trembling, physical weakness and, often, pronounced dyspnea. These are not the usual accompaniments of angina.

If cardiac infarction is suspected in these patients—and it rarely is—it can be eliminated by the measures already enumerated, namely, the absence of fever, leukocytosis, increased sedimentation rate and electrocardiographic changes.

I come now to the condition which, in my own practice, far exceeds all others, perhaps all others combined, as the cause of erroneous diagnoses of cardiac pain, namely, spasm in some portion of the gastrointestinal tract. This may be in the esophagus, at the cardiac end of the stomach, or at the pylorus, but it is my impression that the most frequent location of such spasm is diffusely in the colon, particularly in the transverse colon. Let me add immediately that the great majority of these patients whom I see have not been subjected to careful x-ray study, so that I cannot make that statement with absolute conviction; but it is unimportant, because the point we are now considering is whether the patient has cardiac pain or gastrointestinal pain.

In those patients who are suspected of having cardiac pain, and who are often referred to me with that tentative diagnosis, the major symptom consists of discomfort or actual pain, usually in the left side of the chest, sometimes extending beneath the sternum, but very often not. This discomfort lasts for hours or for days at a time. It is usually not related to physical exertion or emotional excitement, although occasional attacks may begin during walking or mild activity. It sometimes changes its location from day to day. It often has its maximal severity under the breast in women or in corresponding location in men. It often is experienced as high as the clavicle, not infrequently in the anterior axilla, and sometimes in the left side of the back under the angle of the scapula. Even if this discomfort occurs largely or exclusively under the sternum, the features just mentioned should serve to differentiate it from cardiac pain. But there are other factors, also. This pain is seldom relieved by nitro-

glycerin; or if any relief whatsoever is obtained, it is variable and unpredictable. It is usually abolished for long periods by the administration of antispasmodics. Changes in the electrocardiogram indicative of coronary insufficiency or coronary thrombosis are absent unless the patients had those conditions previously. It is very disturbing to reflect upon the large number of patients whose physicians have made confident diagnosis of coronary thrombosis or angina, although the pain has never been beneath the sternum, it has never been associated with physical effort or emotional excitement, and it has never been relieved or prevented by nitroglycerin. If physicians would only keep in mind the characteristic features of cardiac pain, other types of pain could often be excluded within two or three minutes by simple questioning of the patient.

Hiatus hernia of the stomach apparently exists in a very large number of presumably healthy people; in most of them it gives rise to no appreciable or troublesome symptoms, at least, any that are recognized. But in some, apparently, these hernias do give rise to pain that many competent physicians have regarded as of cardiac origin, resembling angina or cardiac infarction. In my own experience, it has almost invariably been the latter, that is, cardiac infarction. But there have been many medical articles stating that the pain of hiatus hernia is sometimes indistinguishable from angina.

I would not be so presumptuous as to say that confusion should never occur, but I think one can say reasonably that it should not occur very often—although, perhaps, my own experience has not been a very satisfactory guide. Perhaps I have just been so unfortunate as not to see many patients with hiatus hernia whose pain was like that of angina. But I do think there are differences in the two conditions that should, within a short time, lead one to the correct diagnosis.

The pain of hiatus hernia occurs most frequently with the patient lying down or leaning forward, and it usually disappears fairly promptly if he stands erect. It usually lasts very much longer than the pain of angina. It may last for a great many minutes or for several hours at a time.

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 81

Nitroglycerin is said to give relief from the pain of hiatus hernia in many cases. Undoubtedly, that is true or appears to be true. Dr. Chester Jones of Boston, whose paper of a few years ago is frequently cited in support of the similarity of angina and hiatus hernia, has emphasized that the use of nitroglycerin will very often point clearly to a diagnosis of hiatus hernia. He says that, occasionally, nitroglycerin will give relief from hernial pain very promptly, and perhaps make one think that the pain is of cardiac origin; but just as frequently, or far more frequently, it will fail to give relief. In any patient, it may give relief one day and the next day have no effect whatsoever. This variable and unpredictable effect of nitroglycerine should make one suspect that the diagnosis is hernia and not angina.

It has also been said that one confusing feature is the fact that patients with hiatus hernia may develop substernal pain on walking, which is certainly true. In my own experience the association has always seemed a fortuitous one but, nevertheless, some patients will say that they get pain in the chest when they walk. Closer questioning will almost invariably reveal that the pain occurs more frequently while at rest or while in bed than it does when walking, and also that most of the time they can walk freely without pain. In other words, the relationship between the pain and walking is a very inconstant and variable one.

Furthermore, one must recognize that any patient may have both angina and hiatus hernia. That is beautifully illustrated by a case described by Dr. Jones. His fourth patient was a man of sixty-five who regularly developed substernal squeezing pain when he walked about a hundred yards. He had a large hiatus hernia. The hernia eventually became so troublesome that it was eliminated by surgical operation. Following his recovery from the operation, the patient continued to develop the same kind and intensity of substernal pain on walking the same distance. In other words, the patient had both angina and hiatus hernia. The pain on walking had nothing whatever to do with his hernia, as shown by the fact that it persisted after the hernia was eliminated.

There are just two other conditions that I wish to mention briefly. The first is arthritis of the upper dorsal or lower cervical spine, a condition that is not uncommon in elderly people—exactly the same groups that are most likely to have cardiac pain. In some of these people, the pain is not experienced at all over the spine, but is felt over the top of the shoulder, along the trapezius ridge, often extending down over the anterior surface of the chest, and into the left arm. A good many of the patients whom I have seen who were suspected of having angina or coronary thrombosis have had that diagnosis based exclusively on those two circumstances, namely, pain in the front of the left chest and radiation of that pain into the arm. Their doctors have not questioned the patients closely enough to learn that the pain is precordial rather than substernal, and that it invariably radiates down the outside of the arm, rather than down the inner aspect. This has been the radiation in every patient I have seen; my neurological and orthopedic friends assure me that their experiences are similar.

Sometimes the pain may extend to involve the substernal area. It usually lasts far longer than anginal pain. It often occurs in patients after they have been lying down or after prolonged sitting in one position. The correct diagnosis may be revealed by finding residual tenderness lasting for some hours over the costochondral junctions, or over the adjacent portions of the ribs. Often the pain in the chest can be reproduced accurately by firm pressure over the upper spine, or by forcible flexion of the patient's head. If cardiac infarction is suspected—and I think it is very seldom—it can be eliminated in the manner already repeatedly indicated, by securing blood counts, sedimentation rates, and electrocardiograms.

In these patients, even more than in those with hiatus hernia, it is important to keep in mind that both angina and arthritis may be present, because both tend to occur with greatest frequency in people who have reached the later decades of life. It may, therefore, be necessary in a given case to have a period of study and observation of several days before one can determine whether the symptoms are due to a single condition or to several.

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 83

Finally, I wish to speak briefly, and with some reluctance, about gallbladder disease. I do so reluctantly because apparently my experience has differed rather sharply from that of my colleagues, and has led me to believe that gallbladder disease seldom gives rise to pain which must be regarded as probably originating from some organ or tissue within the thorax. I know, of course, that gallbladder disease may cause pain which radiates into some part of the chest. Usually it is into the right lower part of the thorax; often it radiates posteriorly to involve the right side of the back, sometimes to the subscapular area, and very often to the top of the right shoulder. That radiation of pain very seldom suggests involvement of the heart.

I have been told by competent internists that the pain sometimes is localized beneath the sternum; but in the few instances where I have had an opportunity to see such patients, careful questioning has revealed that this was not an accurate statement. I have never known any instance in which gallbladder pain radiated into the inner aspect of the left upper arm, although I know it can be produced experimentally in patients who have gallbladder fistulas by forcible distention of the gallbladder. It so happens that I have never observed it in any patient whose pain was conclusively shown to be due to gallbladder disease. But since my practice is largely restricted to patients with cardiovascular disease, it is quite likely that I have not seen enough patients with gallbladder disease to make this statement very meaningful. The history often reveals such conclusive items as typical gallstone colic or jaundice in the past; and physical examination frequently reveals tenderness in the right upper quadrant. The differentiation—the final differentiation—can usually be made on the basis of such things as x-ray study of the gallbladder, electrocardiograms, and observations upon the sedimentation rate and leukocyte count.

No one could be more acutely conscious than I am of the great inadequacy of this discussion. As stated at the beginning, no attempt has been made to consider all the conditions that could give rise to pain in the chest, and a good

many have been deliberately ignored. It seemed wiser to consider even briefly some of the more important causes than to invite your attention to such relatively unimportant ones as fracture of a rib or costal cartilage, herpes zoster and the scalenus anticus syndrome, which are rarely confused with the important conditions.

Even this very sketchy survey indicates that pain arising from the heart as a result of inadequacy of the coronary blood flow, from the pericardium, from the aorta, from the pulmonary artery and mediastinum, may be practically identical in terms of character, location, duration and distribution. The most likely explanation for this striking similarity would seem to be that the pain impulses arising from these various organs and tissues probably travel over the same nerve pathways in order to gain access to the spinal cord, and thence to the sphere of consciousness in the brain.

From the standpoint of life insurance, if there is any lesson to be derived from this modest survey of a very important field, I think perhaps it is the one mentioned by Dr. Harrison. I have taken advantage of your kindness and patience to mention fourteen different conditions, if we consider coronary thrombosis, coronary insufficiency and angina as three rather than one. Of those fourteen conditions it seems to me that at least six or seven are of very little importance in determining whether or not the patient should be accepted for life insurance. Consequently, it becomes of the very greatest importance to insist that a diagnosis of cardiac infarction or of coronary arterial disease should not be made except on the basis of evidence that is beyond doubt, because it places a stigma upon the patient which may interfere seriously with his chances of securing life insurance.

I should like to close by expressing once more my very grateful thanks to Dr. Ylvisaker and Dr. Dieuaide for their most gracious and generous comments, and also to each of you for an extraordinary degree of attentiveness and responsiveness which has given me great pleasure. Thank you.

## DIFFERENTIAL DIAGNOSIS, CHEST PAIN 85

PRESIDENT YLVISAKER—We are all very grateful to both Dr. Dieuaide and Dr. Marvin for their talks here today and for their previous contributions to all our knowledge regarding cardiovascular diseases.

The next speaker on our program needs no introduction. Since 1934, he and his associates, Dr. Kiessling and Miss Lyle, have been analyzing the information they have gathered from the electrocardiograms submitted in connection with applications for insurance to The Prudential, and today they have the results of this study to present to us. Their discussion will be presented by Dr. H. B. Kirkland with this provocative title "Electrocardiography: The Facts of Life."

THE EVALUATION OF CERTAIN FUNDAMENTAL  
ELECTROCARDIOGRAPHIC PATTERNS IN THE  
SELECTION OF INSURANCE RISKS

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Herewith are set forth some of the facts of life in electrocardiography. We three are crusading for the common man, who struggles daily with related underwriting problems without the comforting cushions of high theory and speculation. We deal empirically with patterns which have become associated, over the years, with the normal and abnormal cardiovascular system. We are beset continually by the borderline and the equivocal. We are confronted all too frequently by the results of technical inadequacy. Our opinions are found to be at variance with those of cardiologists of acknowledged eminence, who are not called upon to offer their wherewithal at the prognostic pari-mutuel window. Having no realistic statistics on which to lean, quoting chapter and verse to our embittered field representatives, we are castigated as unworthy members of a specialized and supernumerary species, and suspected as master minds in a gigantic plot to deliver preferred business into the hands of our competitors.

This organization has been honored by many masterly presentations on electrocardiography. The literature is studded with gems of varying brilliance covering the whole gamut of a well worked through but elusive subject. But out of all this we extract only unconvincingly the answers to those simple-appearing but deceptive questions which plague us daily as we strive to correlate the electrocardiographic contours with the clinical background and to produce fair decisions. This paper is an attempt to set forth

some very hard facts about a few of the findings which recur continually in a manner calculated to contract effectively our own expectancies. No theoretical considerations will be evoked. Our foundation is on the bed rock. No claims to final solutions are advanced. We will present our data, hoping that interest will be stimulated and that our conclusions will help in any related future underwriting approach. Nothing that we have to say will obviate the necessity of occasionally consulting that indispensable diagnostic tool, the crystal ball.

#### *Electrocardiographic Impairments Considered*

The underwriting of risks having the findings shown in Table 1 is fraught with continued difficulty, in that they are very common and in that experience as to their significance is either inadequate or conflicting. It should be set forth further that we have not covered electrocardiographic impairments which are so unequivocally abnormal as to eliminate the possibility of extending protection on any basis whatever. We would draw your attention here to the fact that any suggestions as to evaluation for underwriting purposes are based on a range of 0 to plus 400 per cent.

TABLE 1  
ELECTROCARDIOGRAPHIC IMPAIRMENTS  
CONSIDERED

- Premature contractions
- Marked sinus arrhythmia
- Abnormal P waves
- Prolonged auriculo-ventricular conduction
- Prolonged intraventricular conduction
- Normal late activation of conus
- Conus block
- Incomplete right bundle branch block
- Complete right bundle branch block
- Complete left bundle branch block
- Notched T waves
- Low T waves in Leads V5 and V6
- T wave aberrations in Leads aVL and aVF

*Material and Method*

Our material was drawn from a file containing more than 15,000 electrocardiograms on just over 6,000 Home Office employees of the Prudential. Observation began in 1933. Since 1942 every record has included the standard and unipolar extremity leads, as well as precordials V1 through V6. About this same time, our interest in incomplete right bundle branch block led us to add a lead over the right chest routinely. Our exposure periods vary, therefore, in that the study of premature contractions, for instance, was extended back eighteen years, while that of any impairment involving the more recently added leads covers only about half that time.

Since electrocardiograms have never been made routinely at the Prudential, and since our periodic health surveys have always been on a voluntary basis, it follows that our material does not represent a population cross-section. It tends to be weighted with pathology and with the older ages. However, as compensation, we have the advantage of very exact knowledge of the interval history in all except very rare instances. This has enabled us to record with considerable precision not only mortality but the development of morbidity.

The groups to be studied, including the controls, were formed by searching the entire file and assembling every case showing the electrocardiographic finding under consideration, without regard to the clinical situation. The cases so assembled were then tabulated by age and clinical status, special attention being directed to the percentage of cases within each age group in which there was other evidence of cardiovascular involvement. In addition, the cardiovascular deaths were recorded, deaths from unrelated causes being ignored. Note was made of the number of cases, normal clinically at the outset of observation, in which cardiovascular abnormalities, including significant electrocardiographic changes, developed during the follow-up period, but which were still alive at its termination. The over-all distribution by age was also noted.

*Control Group*

The procedure followed in assaying the weight of pathology and age in the file was to form a control group (Table 2) from the same source material to serve as a standard of measurement. This control group was set up in the same way as the other groups, namely, by first assembling the cases showing a specific electrocardiographic feature, in this instance, no abnormalities whatever. The number, 750, was arbitrarily selected as adequate, and these tracings were drawn out alphabetically.

TABLE 2  
CONTROL GROUP, ELECTROCARDIOGRAMS  
NORMAL

750 Cases. Average Duration 3.5 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>			
Clinically Normal	201	0	10
Clinically Abnormal	47 (19%)	0	
<b>40 and over:</b>			
<b>67% of total</b>			
Clinically Normal	339	0	30
Clinically Abnormal	163 (32%)	9	

It should be emphasized that this is not a "normal control group" in the usual sense, but rather a control group designed to demonstrate the extent to which our file is actually weighted with pathology and age. It is evident that this is not the usual statistical approach, but the results will show, we believe, some interesting and valuable trends which should alert us to dangers inherent in some impairments and give us some degree of reassurance in connection with others. It should also be pointed out that, measured in terms of the life span of the average policy, our periods of observation are quite short, and, hence, any conclusions must be somewhat guarded. Nor can the fact be overlooked that, in some in-

stances, longer exposure periods would almost certainly produce a higher percentage of adverse developments and cause the picture to be even more unfavorable.

Because of the inevitable variations in exposure periods of different impairments, it is impossible to compute accurately, on a percentage basis, either morbidity or mortality rates. Table 2 summarizes the situation in our control group. The average duration of observation was 3.5 years. Two-thirds of the cases were aged 40 or over. Of the 248 cases under age 40, 19 per cent showed some evidence of abnormal cardiovascular involvement, most frequently in the form of hypertension or rheumatic valvular disease. Of those normal at the outset, 10, or 5 per cent, developed some abnormality during the follow-up period. Above 40, as one would expect, the percentage of those not normal clinically was higher, 32 per cent. Of those normal clinically, 30, or 9 per cent, experienced later unfavorable developments, including a number of instances in which definitely adverse electrocardiographic features appeared. Among the abnormals, there were nine cardiovascular deaths. The abnormalities in this group were predominantly of hypertensive or arteriosclerotic origin. Attention is drawn once more to the percentages of abnormals in the two age categories, namely, 19 per cent and 32 per cent, and also to the number of normals developing impairments. These will be the focal points of comparison throughout. All tables are arranged in the same format.

#### *Premature Contractions*

Digressing for just a moment before proceeding with comments on this arrhythmia, we wish to express the personal conviction that premature contractions represent as consistently irritating an impairment as any in our rating manual. We have struggled doggedly with the writing and rewriting of rules for action. We have never pleased anyone. Our medical staff, our lay underwriters, and our field representatives are united in the conviction that each revision is worse than what went before. When one speaks of premature beats, how many is many? Suppose they are here today and gone tomorrow? What constitutes a significant increase after

exercise? Or, if they do not disappear after exercise, how is a decrease to be evaluated? We have discovered that the only way to handle one of these critics is to admit freely that the rules are dreadful, and to suggest that he or she draw up a new set. We are still waiting.

We spent more weary hours than we like to remember classifying and tabulating all of these findings, trying valiantly to find some satisfactory method by which ectopic beats could be broken down according to number. It just could not be done. There was astonishingly little difference when a separation was made between "few" and "many," ten per minute being the dividing line. Since premature beats characteristically vary so greatly in frequency from day to day, and even from minute to minute, there seems little justification for classifying them on the basis of a fortuitous count at the time of examination, although, from a practical standpoint, some numerical breakdown may be deemed necessary. Little difference was noted between premature contractions of supraventricular and ventricular origin.

Table 3 deals with unifocal supraventricular premature beats. As compared with the control group, more were in the upper ages, and a substantially greater number, at all

TABLE 3  
UNIFOCAL PREMATURE BEATS,  
SUPRAVENTRICULAR

237 Cases. Average Duration 4.9 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>			
Clinically Normal	39 (a)	0	2
Clinically Abnormal	19 (33%) (b)	1	
<b>40 and over:</b>			
<b>76% of total</b>			
Clinically Normal	51 (c)	0	7
Clinically Abnormal	128 (72%) (d)	36	
Developed multifocal premature beats:			
	(a) 1; (b) 1; (c) 2; (d) 19.		

ages, showed other cardiovascular abnormalities. The number of cardiovascular deaths was striking.

Unifocal ventricular premature beats (Table 4) showed a very similar trend.

TABLE 4  
UNIFOCAL PREMATURE BEATS, VENTRICULAR  
476 Cases. Average Duration 5.2 Years

		Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>				
Clinically Normal	88 (a)		0	5
Clinically Abnormal	42 (32%) (b)		2	
<b>40 and over:</b>				
<b>73% of total</b>				
Clinically Normal	102 (c)		3	14
Clinically Abnormal	244 (71%) (d)		71	
Developed multifocal premature beats:				
	(a) 4; (b) 5; (c) 8; (d) 19.			

There was a combined total of 713 cases (Table 5). Recalling that the control group showed only 19 per cent abnormal clinically under 40, and 32 per cent in the upper age group,

TABLE 5  
UNIFOCAL PREMATURE BEATS,  
SUPRAVENTRICULAR OR VENTRICULAR  
713 Cases. Average Duration 5.1 Years

		Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>				
Clinically Normal	127 (a)		0	7
Clinically Abnormal	61 (32%) (b)		3	
<b>40 and over:</b>				
<b>74% of total</b>				
Clinically Normal	153 (c)		3	21
Clinically Abnormal	372 (71%) (d)		107	
Developed multifocal premature beats:				
	(a) 5; (b) 6; (c) 10; (d) 38.			

it is difficult to see how one can disregard so marked a variation in the incidence of organic involvement, even after adjustment is made for the difference in duration between the two groups. It would seem reasonable to continue to assess these cases conservatively, and to look with very considerable suspicion on individuals presenting a combination of this impairment with other cardiovascular findings.

It is quite likely that, especially at relatively young ages, knowledge that premature contractions have been present over a long period of time may permit more lenient treatment. Our investigation did not yield any satisfactory answer. It is worth noting, however, that of the 127 persons under 40 and normal clinically, 5 developed multifocal ectopic beats; of the 61 abnormal clinically at these ages, 6 did similarly; of the 153 and 372 aged 40 and over, 10 and 38, respectively, eventually showed multiple foci. The adverse significance of this sequence of events is well demonstrated in Tables 6, 7, and 8.

The 80 cases showing multifocal ectopic beats, supraventricular and ventricular (Table 6), had an age distribution similar to that of the unifocal group. The high incidence of abnormal cardiovascular abnormalities otherwise is evident.

TABLE 6  
MULTIFOCAL PREMATURE BEATS,  
SUPRAVENTRICULAR AND VENTRICULAR  
80 Cases. Average Duration 4.8 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>			
Clinically Normal	8	0	0
Clinically Abnormal	10 (56%)	0	
<b>40 and over:</b>			
<b>78% of total</b>			
Clinically Normal	12	0	2
Clinically Abnormal	50 (81%)	12	

Table 7 shows the situation among those individuals having multifocal ventricular premature beats. There is no significant difference in comparison with the group included in Table 6, and a summarizing table combining all multifocal ectopic beats is useful in pointing up conclusions (Table 8).

TABLE 7  
MULTIFOCAL PREMATURE BEATS,  
VENTRICULAR

41 Cases.	Average Duration	6.2 Years	
	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>			
Clinically Normal	4	0	1
Clinically Abnormal	6 (60%)	1	
<b>40 and over:</b>			
<b>76% of total</b>			
Clinically Normal	3	0	2
Clinically Abnormal	28 (90%)	14	

TABLE 8  
MULTIFOCAL PREMATURE BEATS,  
SUPRAVENTRICULAR AND/OR VENTRICULAR

121 Cases.	Average Duration	5.3 Years	
	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under age 40</b>			
Clinically Normal	12	0	1
Clinically Abnormal	16 (57%)	1	
<b>40 and over:</b>			
<b>77% of total</b>			
Clinically Normal	15	0	4
Clinically Abnormal	78 (84%)	26	

These cases are, of course, relatively few in number, but the percentages of abnormals clinically is extremely high when compared with those of the control group, 19 per cent and 32 per cent, and materially higher than those in the unifocal group.

An interesting point to be noted here is that when premature beats, either unifocal or multifocal, developed in the course of long-standing cardiovascular impairments such as hypertensive, arteriosclerotic, or rheumatic heart disease, the individual did not, as a rule, live very long thereafter.

There is strongly suggestive evidence that premature contractions are not always quite the innocent findings that many of us have considered them to be. Their presence certainly indicates the necessity of a very careful survey of the entire cardiovascular background, and we feel that even the occurrence of small numbers, especially in the upper age groups, justifies insistence on an electrocardiogram. We can be certain that the co-existence of this irregularity with other abnormal cardiovascular manifestations requires a most cautious approach. We are confident that the appearance of premature contractions in individuals otherwise impaired from a cardiovascular standpoint is an extremely grave sign indeed. By inference, their appearance in an older person who has never been known to show them before should also constitute a danger signal. On the credit side, it is probable that the known presence of ectopic beats over a period of years is a less unfavorable observation. We certainly have no evidence to justify our being unduly conservative in dealing with younger individuals showing premature contractions who are otherwise entirely normal.

The foregoing suggests that considerably more care should be exercised than in the past in ruling out the presence of some underlying abnormality.

#### *Marked Sinus Arrhythmia at the Upper Ages*

We now come to an enthusiastically neglected subject, marked sinus arrhythmia in upper age individuals. Figure 1 illustrates what we mean. This is a continuous strip of a bipolar sternal lead designed to bring out the P waves. The record shows the abruptness and degree of the change in auricular timing, the rate varying from 48 to 97. In none of our cases was there any significant change in P wave contours which could be construed to indicate the presence of

an ectopic focus, nor was any correlation with respiration evident.

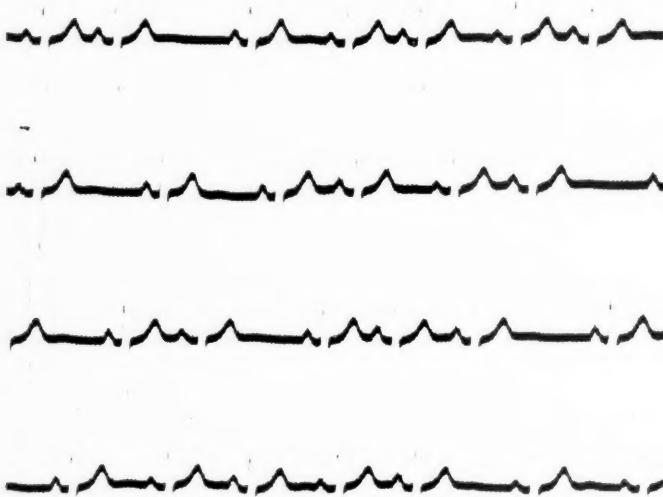


Figure 1

There were only 18 cases (Table 9) which satisfied our strict criteria. There were only two normal clinically, and one of these developed significant electrocardiographic aberrations during the period of observation. There were 6 deaths among those clinically abnormal at the outset.

TABLE 9  
MARKED SINUS ARRHYTHMIA OVER AGE 55

18 Cases. Average Duration 5.4 Years

Over Age 55		Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
Clinically Normal	2	0	0	1
Clinically Abnormal	16 (89%)	6		

Even with so small a group, the conclusions seem evident. We infrequently find so marked a degree of sinus arrhythmia among our older applicants, but it would seem that we are justified in subjecting individuals showing even moderate degrees of this arrhythmia to searching cardiovascular scrutiny, and we personally would not hesitate to recommend a substantially substandard classification even if the case were otherwise negative. The view of this impairment reflected in our analysis is supported by a number of clinical opinions which have appeared variously, although briefly, in the literature.

#### *Abnormal P Waves*

It would be expected that this group would be heavily weighted with pathology, but the figures are nevertheless surprising in demonstrating how relatively infrequently notched, wide, and high P waves are encountered in the absence of clinical cardiovascular involvement.

Wide P waves were defined as those exceeding .11 sec. and high P waves as those with a positive amplitude of more than .25 mv. Table 10 demonstrates the preponderance of abnormal clinical backgrounds, with a heavy related death ratio. Well over half of the cases were found in the younger

TABLE 10  
P WAVES NOTCHED, WIDE, HIGH  
132 Cases. Average Duration 6.8 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	31	1	2
Clinically Abnormal	47 (60%)	9	
<b>40 and over:</b>			
<b>41% of total</b>			
Clinically Normal	11	1	2
Clinically Abnormal	43 (80%)	13	

age group, reflecting the high incidence of rheumatic heart disease.

#### *Prolonged P-R Interval*

Delayed auriculo-ventricular conduction was found in 119 of our cases, and the group cannot be considered satisfactory for purposes of evaluation, chiefly because individual cases showed disconcerting variations in the P-R intervals, and it was therefore virtually impossible to break down the degrees of block. Furthermore, in order to assemble any worthwhile numbers, it was necessary to include cases showing P-R intervals of .21-.22 sec., and, with rates of about 75 or under, it is highly questionable whether these intervals represent a real block.

It is not considered advisable to present any figures dealing with this electrocardiographic impairment, in that further observation of an expanding series will be necessary to adequate conclusions. Suffice it to say at this point that, in general, the group showed a rather alarming percentage of clinically abnormal cases. It would certainly seem advisable to exercise every caution in ruling out some underlying pathological process when confronted with delayed A-V conduction, but, when a case is negative otherwise, our series produced no evidence to support an overly conservative underwriting attitude.

#### *Prolonged Intraventricular Conduction*

Prolongation of the intraventricular conduction time takes two broad forms, both illustrated in Figure 2.

In the first two examples (Cases 1 and 2) the QRS contours are quite conventional, but the duration is definitely more than .10 sec. Case 1 shows well defined end points; those in Case 2 are poorly defined. In the third example (Case 3) the contours are bizarre, that is, coarsely notched or otherwise distorted to a degree which attracts attention on first glance at the tracing. In several cases, this bizarre appearance was quite striking, but the QRS duration did not exceed .10 sec.

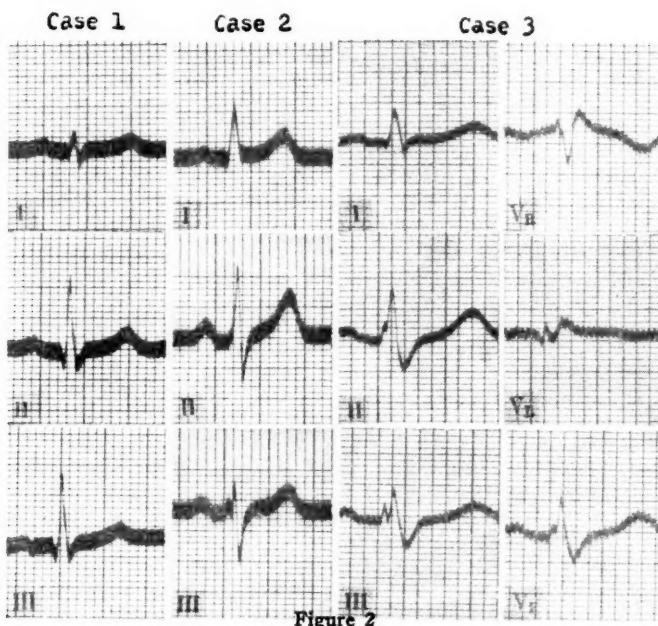


Figure 2

The group embracing the normal contours (Table 11), although small, is a very favorable one.

TABLE 11  
QRS > .10 SEC. WITH NORMAL CONTOUR  
26 Cases. Average Duration 5.3 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	11	0	0
Clinically Abnormal	1 (8%)	0	
<b>40 and over:</b>			
<b>54% of total</b>			
Clinically Normal	10	1	0
Clinically Abnormal	4 (29%)	0	

The bizarre contours, on the contrary, were found in an extremely unfavorable group clinically, with ten deaths among the 15 abnormals aged 40 and over (Table 12).

TABLE 12  
QRS NORMAL OR PROLONGED WITH  
BIZARRE CONTOUR

28 Cases. Average Duration 3.7 Years

		Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>				
Clinically Normal	3	0	0	0
Clinically Abnormal	5 (63%)	1		
<b>40 and over:</b>				
<b>71% of total</b>				
Clinically Normal	5	0	1	
Clinically Abnormal	15 (75%)	10		

The evidence provided by these cases suggests, therefore, that we need have little concern if wide QRS complexes have entirely normal patterns; in fact, if the clinical background is clear, it would seem that no debit whatever would be called for. In the presence of bizarre notching, however, an extremely conservative stand appears justified.

#### *Conus Patterns*

With the commendable increase in the tendency to include multiple precordial leads in routine electrocardiograms, a peculiar QRS configuration in Lead V2, unaccompanied by any other electrocardiographic aberrations, has been not infrequently noted. This is characterized by a prominent terminal upward QRS deflection, which is less clearly shown, if at all, in Leads V1 and V3. The pattern is best shown one interspace above the usual level of precordial exploration. It is characteristically absent one interspace below the usual level. All of our cases have had chest exploration at three levels, from axilla to axilla. In this manner, we have been able to establish or to rule out the presence of the most common concomitant finding, incomplete right bundle branch block.

Conus block is understood, for the purpose of this analysis, to be a delay in the spread of the electrical impulse to the base of the heart, in the region of the conus.

Figure 3 shows Leads V<sub>3R</sub> through V<sub>3</sub> at the upper, usual, and lower levels in a case having what we believe to be normal late activation of the conus. It will be noted that the QRS duration is not increased in Lead V<sub>2</sub>, and that the QRS contour is not bizarre.

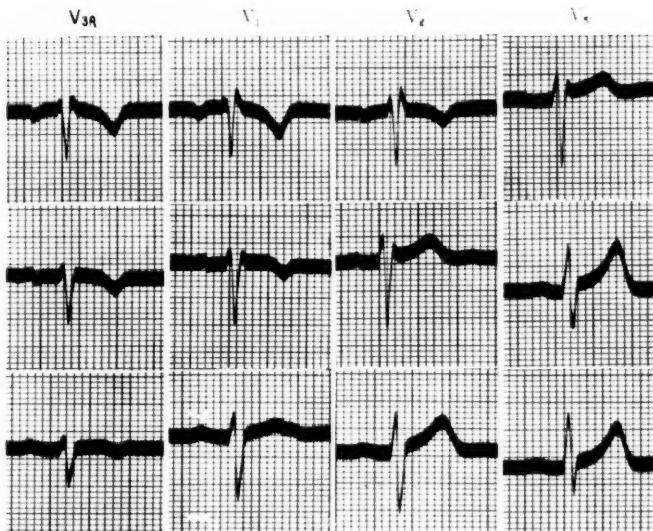


Figure 3

Figure 4 shows the same leads at the same three levels in a case showing wide and distorted QRS complexes in Lead V<sub>2</sub> at the upper and usual levels. This we interpret as representing conus block.

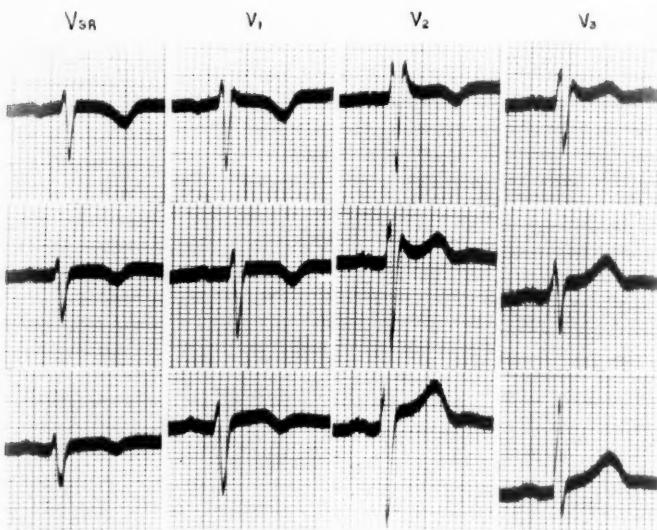


Figure 4

The apparently benign nature of this pattern seems to be borne out by the figures in Tables 13 and 14.

TABLE 13  
NORMAL LATE ACTIVATION OF CONUS  
80 Cases. Average Duration 6.9 Years

		Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>				
Clinically Normal	34	0	3	
Clinically Abnormal	8 (19%)	0		
<b>40 and over:</b>				
<b>48% of total</b>				
Clinically Normal	23	0	6	
Clinically Abnormal	15 (39%)	4		

In what we consider normal late activation of the conus, a substantial number of cases showed percentages of abnormal

clinical involvement strikingly similar to those of the control group.

Bizarre conus complexes, relatively rare, appeared to have no less favorable an outlook, as shown in the few cases summarized in Table 14.

TABLE 14  
CONUS BLOCK, WIDE AND/OR BIZARRE  
19 Cases. Average Duration 5.6 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	11	0	0
Clinically Abnormal	2 (15%)	0	
<b>40 and over:</b>			
<b>32% of total</b>			
Clinically Normal	5	0	1
Clinically Abnormal	1 (17%)	1	

It is to be concluded, therefore, that conus block and normal late conus activation can be considered, in general, benign phenomena.

#### *Incomplete Right Bundle Branch Block*

Incomplete right bundle branch block has been an especial hobby of ours. It would be instructive to show more of our cases, but we can only try to define the pattern, pointing out salient features by a very few illustrations, and demonstrate our experience with a sizable series. The diagnosis has been established in every case by extensive exploration of the chest, from axilla to axilla at three levels, as outlined under conus block. Regardless of the findings in the routine leads, including V4r and V1, precise diagnosis requires demonstration of the characteristic QRS pattern at all levels, this being best shown, usually, in V3r and V4r. We have encountered a number of cases in which our suspicions have been aroused by the findings on the routine tracing, but in which leads made over the right chest failed to corroborate the diagnosis of a significant conduction defect, in that clear-cut S waves terminated the QRS complexes at the lower

level. We believe that a terminal upward QRS deflection or late notching at the lower level is essential to the diagnosis.

Figures 5 and 6 show one example of incomplete right bundle branch block. This is the variety characterized by a late secondary upward QRS deflection in Lead V<sub>1</sub>; the wide, slurred S waves in Leads I, aVL, V<sub>5</sub>, and V<sub>6</sub>, and the prominent terminal upward QRS deflection in Lead aVR are well shown. In general, we diagnose right bundle branch block as complete at and above a QRS duration of .12 sec., although a few cases in which the duration is .12 sec. have been included in the incomplete group because the QRS contours were not those typical of complete block. We wish to emphasize that an increased QRS duration is not essential to the diagnosis: 80 per cent of our cases had a QRS interval measuring .10 sec. or less, and a number measured only .08 sec.

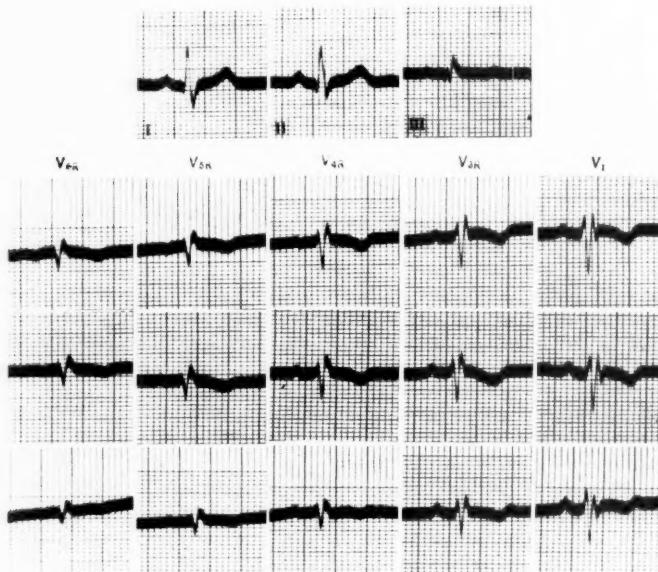


Figure 5

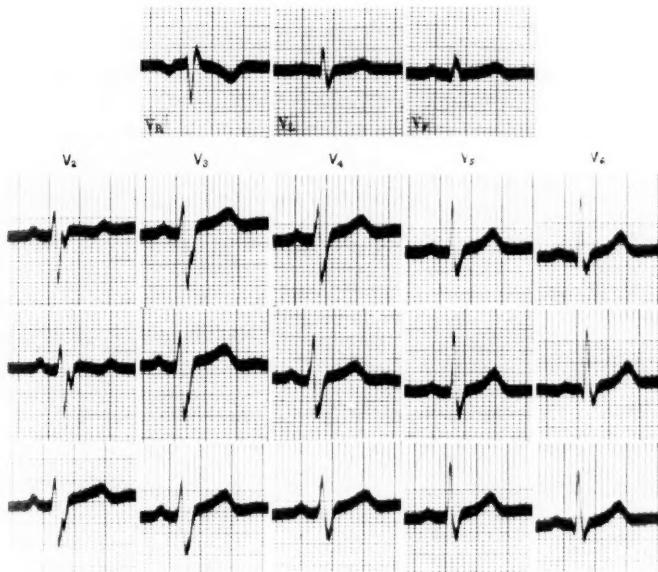


Figure 6

The second example (Figures 7 and 8) shows coarse notching of the ascending limb of S near the iso-electric line in Lead V1. In some instances this notch may be lower down, but it is always in the second half of the QRS complex. The contours in Leads I, aVR, aVL, and in the left precordial leads are the same as those noted in the first example.

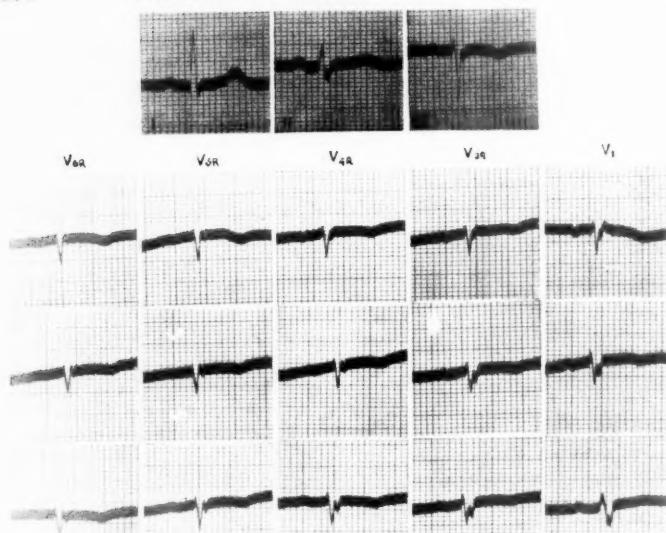


Figure 7

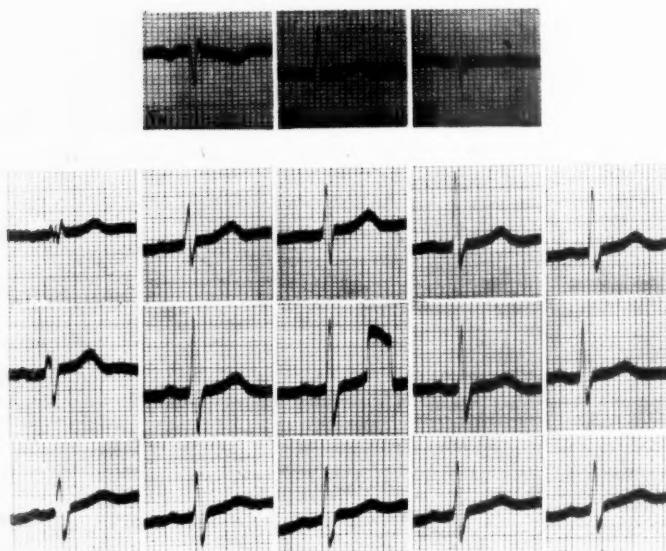


Figure 8

Both of the illustrated types (Table 15) are included in our series of 123 cases, which have an average duration of 6.6 years. Each one of these has had a three level record. These cases were reasonably evenly divided by age, 58 per cent being 40 or over, slightly less than in the control group. Twenty-one per cent of the individuals under 40 had evidence of cardiovascular involvement otherwise, as against 19 per cent in our control group. Thirty-nine per cent of those 40 and over had definite cardiovascular impairments, whereas the related control group showed 32 per cent, hardly a significant difference. As in the control group, relatively few of those originally normal developed definite cardiac abnormalities.

TABLE 15  
INCOMPLETE RIGHT BUNDLE BRANCH BLOCK  
123 Cases. Average Duration 6.6 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	41	0	4
Clinically Abnormal	11 (21%)	0	
<b>40 and over:</b>			
<b>58% of total</b>			
Clinically Normal	43	1	4
Clinically Abnormal	28 (39%)	2	

The foregoing would certainly tend to suggest that we were dealing with a relatively benign process in incomplete right bundle branch block. It has been the feeling in some quarters, and among ourselves, that experience with this pattern was too limited to justify classifying it as eligible for consideration on a standard basis, although debits have, as a rule, been small. We now feel that liberalization of thought is warranted, especially if the background is otherwise negative, and more certainly still if we have the good fortune to have two or more records which show stability over a significant period of time. The usual caution will have to be used in appraising upper age group individuals without previous trac-

ings, but even here our experience would seem to make possible elimination of a debit in clean cases. Associated with other cardiovascular abnormalities, especially hypertension, we are of the opinion that some debit would be advisable in addition to that called for by the clinical impairment involved.

There is undoubtedly some element of danger in a few instances, but these are probably not numerous enough to warrant any alteration in the generally favorable viewpoint implied before. As a warning, however, we set forth three tracings of an individual who, during a period of 10 years, progressed from a normal pattern through incomplete to complete right bundle branch block. Altogether there have been five instances of this type among our 123 cases.

Figure 9 is a tracing made when the subject was 47. It was his first electrocardiogram. The blood pressure was somewhat elevated, but otherwise the clinical background was negative.

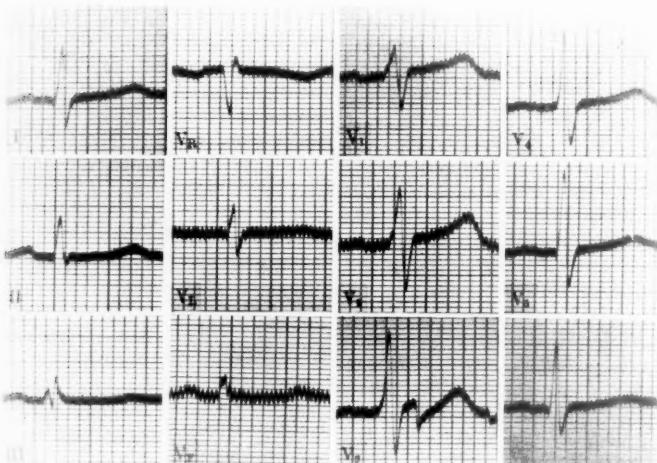


Figure 9

The pattern of Figures 10 and 11, showing incomplete right bundle branch block, appeared five years later, by which

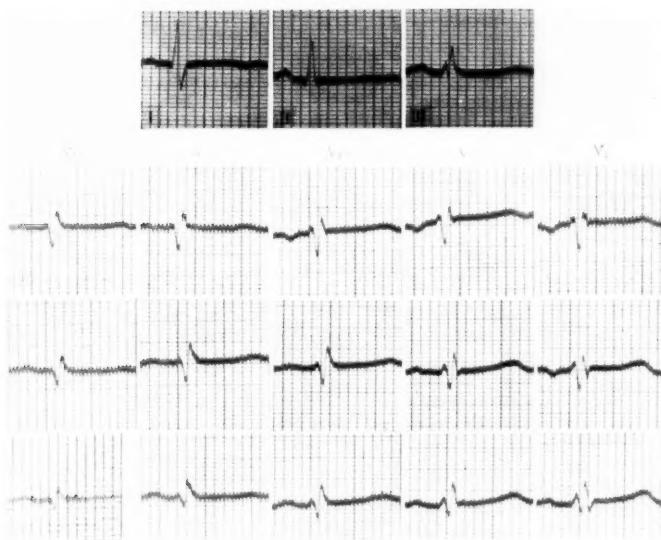


Figure 10

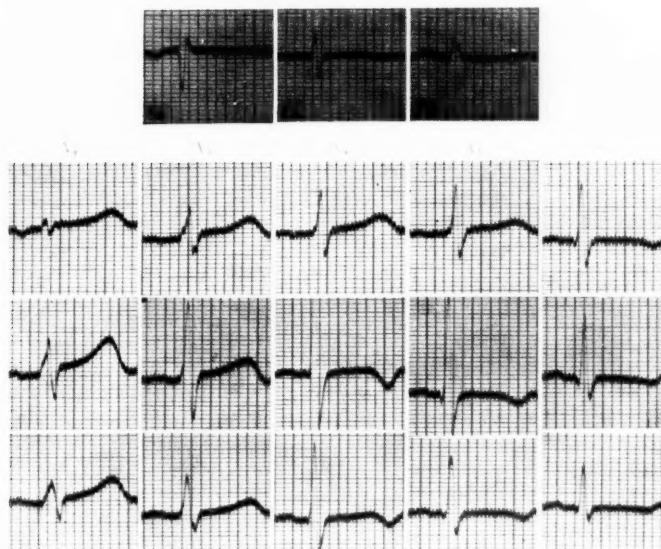


Figure 11

time the subject had an anginal syndrome, showed marked hypertension, and had developed a systolic murmur at the apex. In the interim, diabetes had been discovered.

After a further six months, a record, not shown here, demonstrated the presence of complexes characteristic of both incomplete and complete block.

Figures 12 and 13 show complete right bundle branch block, established shortly thereafter. It is to be noted that the beginning of the QRS complex is the same in incomplete as in complete right bundle branch block in any given electrode position. The subject died, age 57, of arteriosclerotic heart disease, ten years after the initial observation, and four years after complete block became fixed.

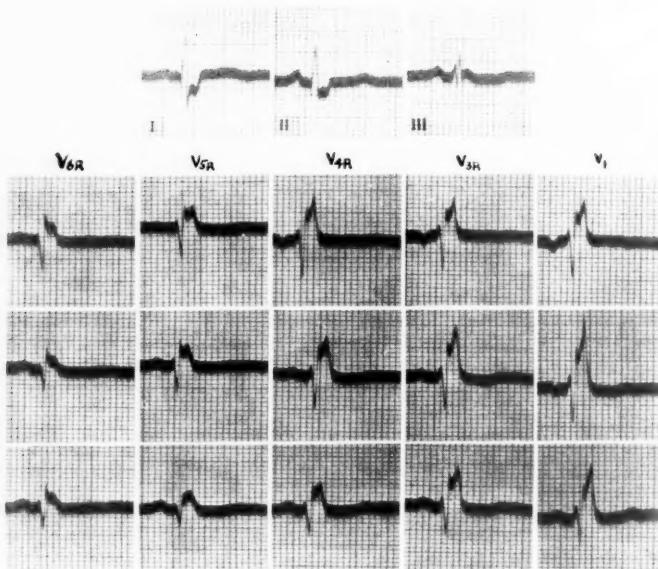


Figure 12

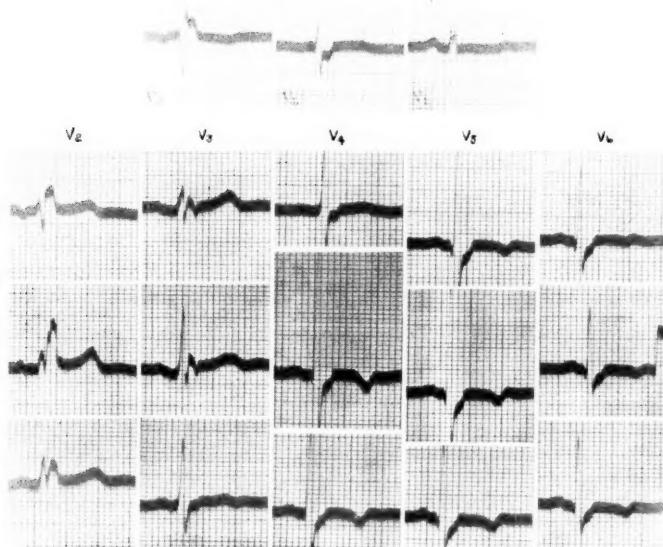


Figure 13

#### *Complete Bundle Branch Block*

Complete bundle branch block is touched on only briefly here, in view of its generally accepted dubious prognosis and more especially because of the relatively few cases in our files. Our experience is adequately summarized in Tables 16 and 17.

It would appear from Table 16 that very considerable hazards would attend any underwriting approach to complete right bundle branch block. It is difficult to see how one could identify enough of the favorable cases to make even a very high substandard offer a sound investment.

The picture of complete left bundle branch block, outlined in Table 17, is even more foreboding. No further comment would seem called for in this connection.

TABLE 16  
COMPLETE RIGHT BUNDLE BRANCH BLOCK

38 Cases. Average Duration 6.8 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	1	0	0
Clinically Abnormal	3 (75%)	0	
<b>40 and over:</b>			
<b>89% of total</b>			
Clinically Normal	13	0	4
Clinically Abnormal	21 (62%)	6	

TABLE 17  
COMPLETE LEFT BUNDLE BRANCH BLOCK

26 Cases. Average Duration 5.9 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	0	0	0
Clinically Abnormal	1 (100%)	0	
<b>40 and over:</b>			
<b>96% of total</b>			
Clinically Normal	4	1	1
Clinically Abnormal	21 (84%)	9	

#### *Notched T Waves*

We have long suspected that the significance of notched T waves is virtually identical with that of inverted T waves in the same leads. We have considered significant only those in which the notching was sufficiently coarse to be unequivocal, occurring in Leads I or II, the unipolar extremity leads, or the precordial leads, except as a transitional phenomenon in Lead V2, as illustrated in Figure 14.

Figure 15 shows the relationship of notched to inverted T waves in a transient phase occurring during recovery from

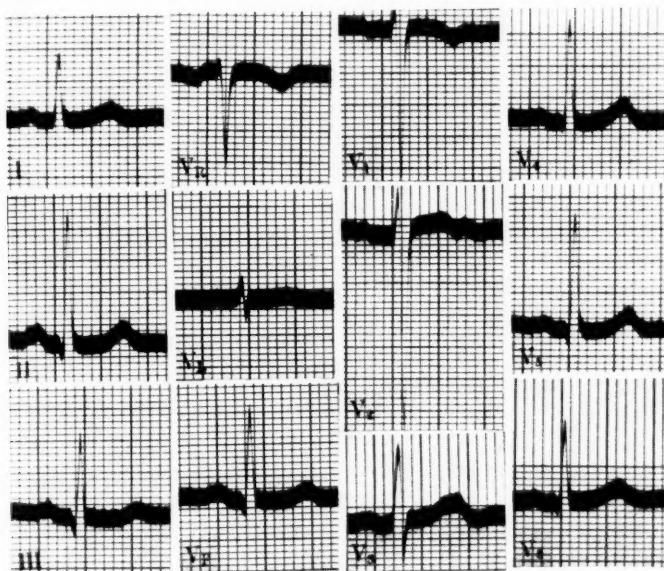


Figure 14

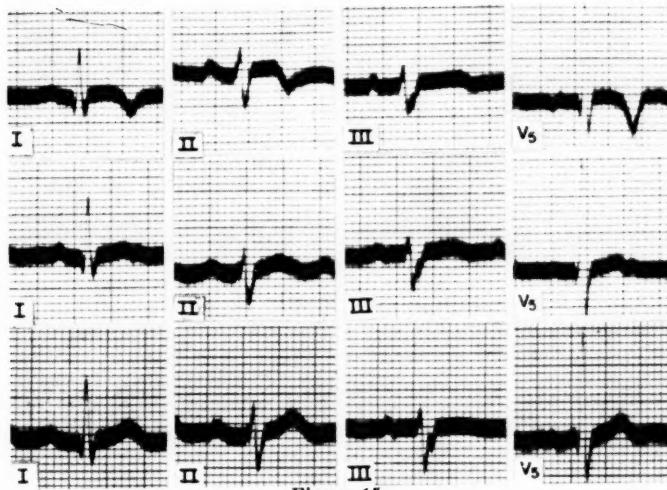


Figure 15

scarlet fever. The first tracing was made when the subject was acutely ill, the second during convalescence, and the third after recovery.

Figures 16 through 18 show the evolution of residual T wave notching at the termination of 14 months' observation of an individual in his forties presenting the pattern of Figure 16 initially.

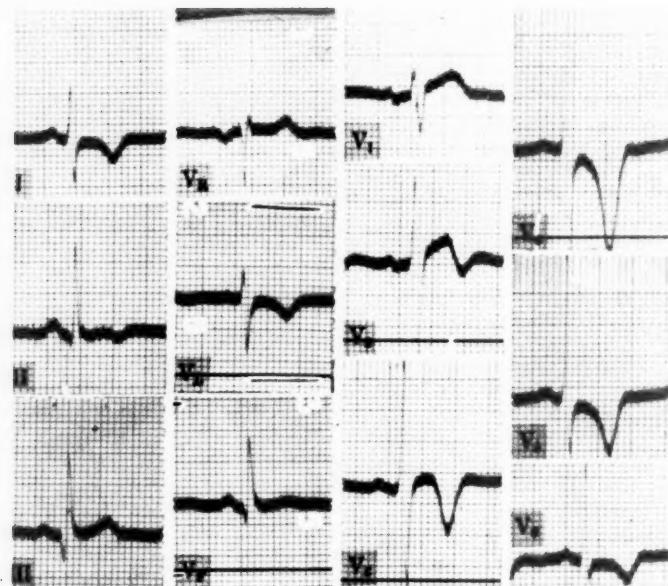


Figure 16

Figure 17 illustrates the over-all appearance of the last record; it is quite evident that, without knowledge of what had gone before, one would not suspect so extensive prior involvement.

Figure 18 summarizes the most significant precordial lead findings. The residual T wave notching would have been the only clue to the underlying pathologic process had no earlier tracing been available.

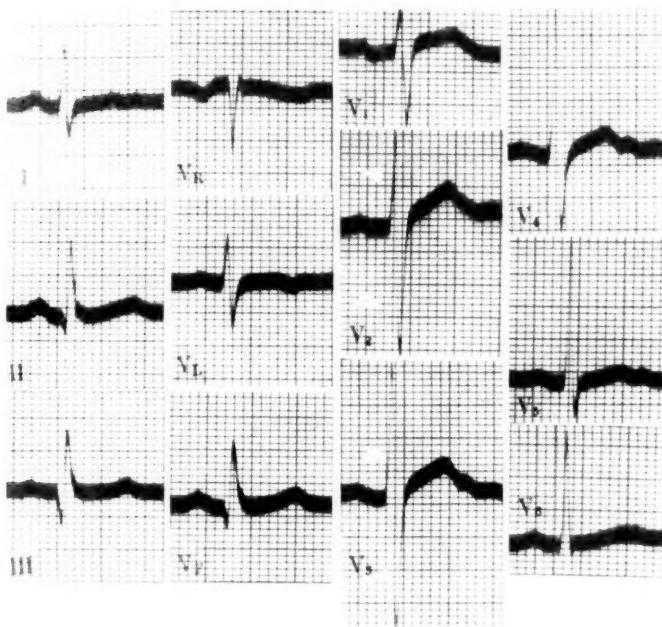


Figure 17

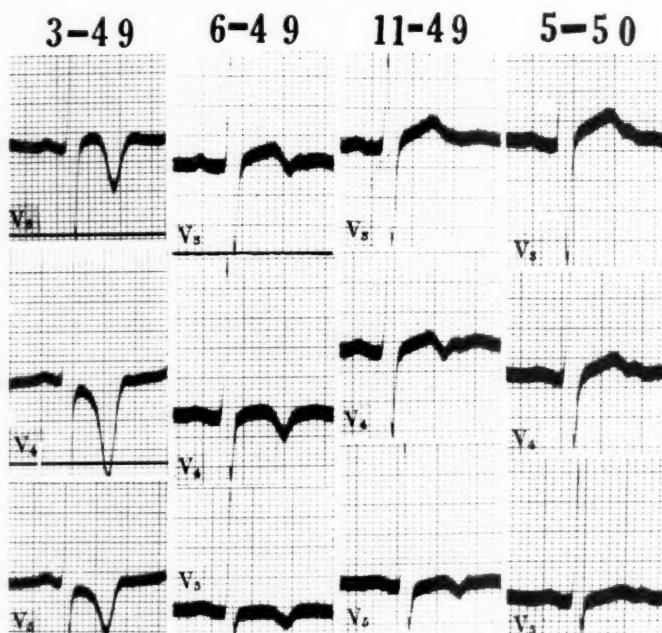


Figure 18

Figure 19 illustrates the converse sequence of events. This man was 62 years of age. Here we have T wave notching in Lead V5 in July, 1947; clinical angina had developed by September, 1947; the pattern of extensive infarction was well shown in November, 1947.

Figures 20 through 22 illustrate the transient appearance of T wave notching coincident with definite clinically observed angina pectoris.

Figure 20 shows a normal tracing.

ELECTROCARDIOGRAPHIC PATTERNS 117

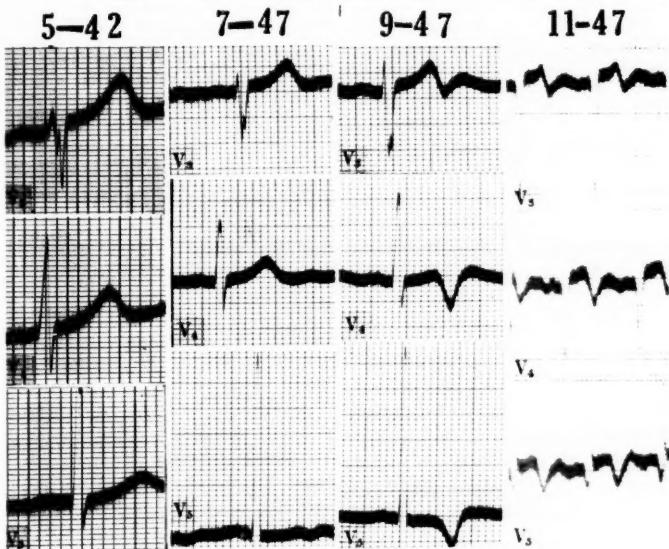


Figure 19

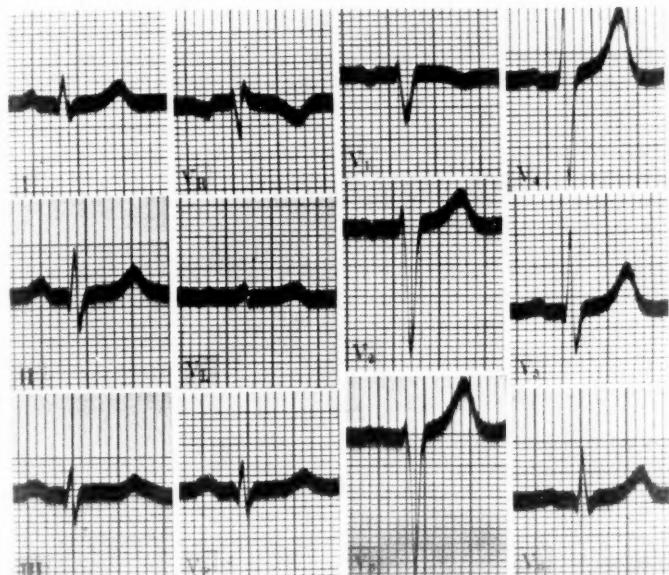


Figure 20

Figure 21 shows a record made when the subject was complaining of substernal pain on exertion; the T wave notching in V4 is evident. This is probably a marginal phenomenon, as the T waves in Lead aVL are inverted.



Figure 21

Figure 22 again demonstrates an entirely normal pattern.

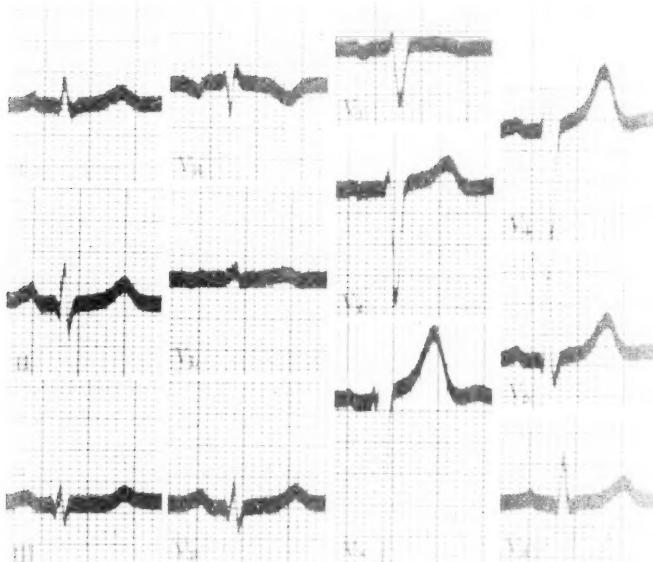


Figure 22

Figure 23 shows notched T waves in one of the precordial leads, with inverted T waves in electrode positions to the left of it and upright T waves to the right. When an electrocardiogram which includes only a single apical precordial lead shows the T waves notched in that lead, it should be borne in mind that a full set of precordial leads might have shown some abnormal inversion to its left or right.

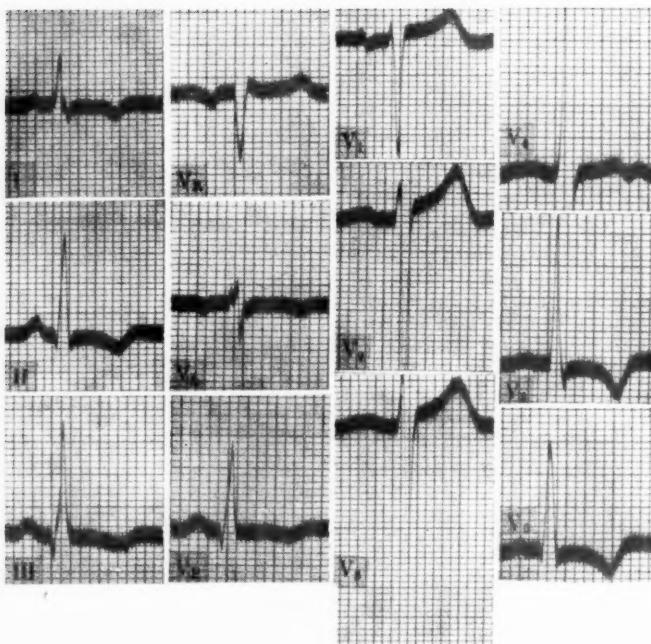


Figure 23

Figure 24 shows, in the first two cases, T wave notching appearing after ventricular premature contractions. The third case in this illustration shows T wave notching, present elsewhere throughout the lead, disappearing after the ectopic beat.



Figure 24

Bearing in mind the fact that we are not including the transitional notched T waves occasionally seen in Lead V2 or, more rarely, in V3, which we found a very favorable group, one sees from Table 18 that individuals showing

TABLE 18  
NOTCHED T WAVES, ISOLATED FINDING  
58 Cases. Average Duration 4.1 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	6	0	1
Clinically Abnormal	5 (45%)	0	
<b>40 and over:</b>			
<b>81% of total</b>			
Clinically Normal	11	0	3
Clinically Abnormal	36 (77%)	4	

significant notching of T waves compare very unfavorably with those in our control group. There were 58 cases, with an average duration of 4.1 years. Most of the cases were aged 40 or over, and 77 per cent of these presented an abnormal cardiovascular background, as against only 32 per cent in the control group. It is to be noted that three of the clinically normal subjects subsequently developed cardiovascular abnormalities, and that there were four deaths among the 36 initially abnormal individuals.

We had 84 cases in which notched T waves were accompanied by other abnormalities in the same electrocardiogram (Table 19). Here the average duration happened to be quite short, 2.7 years, but again the picture is an unfavorable one. A similar high percentage of the upper age subjects had other stigmata of cardiovascular involvement, 5 of 14 normals developed abnormalities, and there were six deaths in the clinically abnormal group.

TABLE 19  
NOTCHED T WAVES WITH OTHER  
ABNORMALITIES IN SAME RECORD

84 Cases. Average Duration 2.7 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	3	0	2
Clinically Abnormal	11 (79%)	1	
<b>40 and over:</b>			
<b>83% of total</b>			
Clinically Normal	14	0	5
Clinically Abnormal	56 (80%)	6	

We are convinced, on the basis of our survey of these 142 cases, that notched T waves deserve to be held in high respect when they confront us on a selection electrocardiogram. There is an unusually large proportion of cases in the upper age group; a high percentage of cases shows other abnormalities; contrasted with the control group, a signif-

icantly higher percentage later developed cardiovascular abnormalities, and the relative number of deaths was high. It is our feeling that if the finding is unequivocal, that is, occurring in one of the leads mentioned and definitely not due to any technical aberration, and if there is in the clinical background anything else suggestive of cardiovascular involvement, the applicant concerned is uninsurable. If the T wave notching appears as an isolated phenomenon in a case which is otherwise entirely clean, we believe that we are dealing with a very considerably contracted group expectancy. As in any such situation, a substantial rating would seem essential. We are in very great doubt as to whether individuals showing significant notched T waves deserve to be placed in a category much better than those who have recovered from an acute infarction. Some of them will do very well, but too many will not—too many to justify much in the way of underwriting liberality.

#### *Low T Waves in Leads V5 and V6*

We will touch only briefly on a small but important series of cases, those showing T waves positively directed but less than 1 mm. in amplitude, with full standardization, in Leads V6 or V5 and V6 (Table 20). The records concerned showed no other questionable findings. We had 42 cases, with an average duration of 3.9 years, and almost all were aged 40 or over. The adverse nature of this pattern is pointed up by a percentage of clinically abnormals of 75, whereas the control group had only 32 per cent. Two of ten clinically normal at the outset developed cardiovascular abnormalities, and there were three deaths among the 30 clinically abnormal initially. It is interesting, also, that we found so few cases in which this was an isolated abnormality; several times this number of cases showed low T waves in V5 and V6, but could not be included in our series because there were other abnormalities in the records.

This group is too small to warrant fixed conclusions, but the trend is evident, and we feel that extreme underwriting caution is called for when we are faced with these contours.

TABLE 20

LOW T WAVES (< 1 mm.) IN V6 OR IN V5  
AND V6, ISOLATED FINDING

42 Cases. Average Duration 3.9 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	1	0	0
Clinically Abnormal	1 (50%)	0	
<b>40 and over:</b>			
<b>95% of total</b>			
Clinically Normal	10	0	2
Clinically Abnormal	30 (75%)	3	

It is highly unlikely that we would consider on any basis a combination of the pattern in question with any other significant cardiovascular abnormality. When the finding is not associated with clinical abnormalities, which is not often, we feel that a moderate rating would be in order, but we need a little more experience with this series before reaching final conclusions.

*T Wave Aberrations in Leads aVL and aVF*

The final category in this discussion involves abnormal or questionable T waves in Leads aVL and aVF, T waves elsewhere throughout each record being absolutely normal.

Figure 25 presents a record showing, as an isolated finding, a diphasic T wave in Lead aVF with a predominantly positive QRS; a similar type of contour appears frequently in Lead aVL.

Figure 26 illustrates the various types of unipolar extremity lead T wave patterns which we have investigated at this time. The first row shows aVL patterns from five different cases, the second row corresponding aVF patterns from five other cases. The variations in QRS and T contours are evident. In all of our cases, T waves elsewhere throughout the record were unequivocally normal.

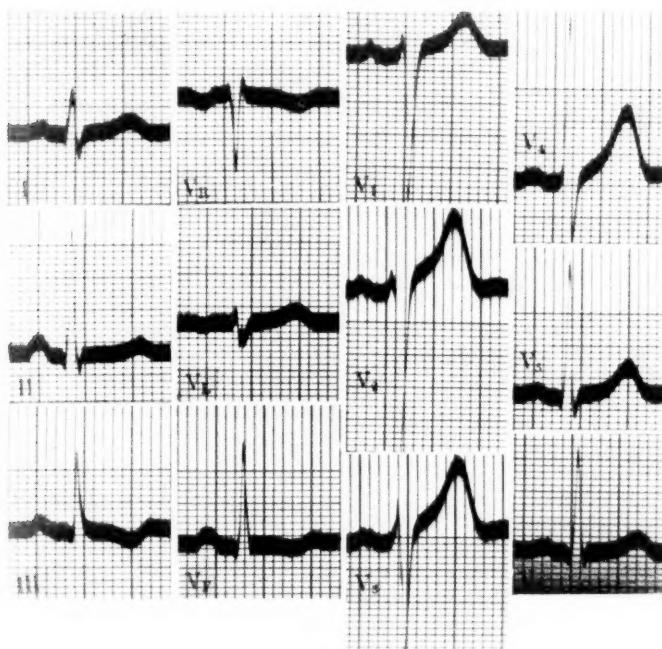


Figure 25

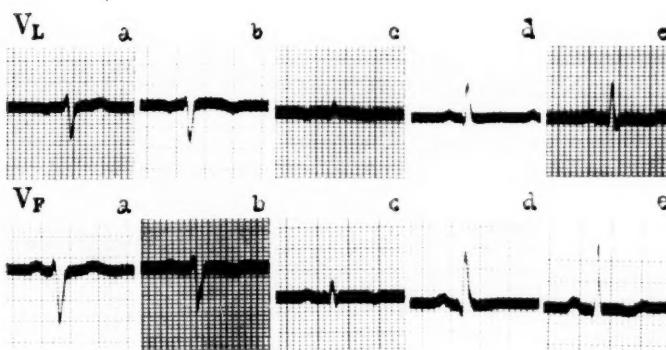


Figure 26

First of all, we confirmed our impression that T wave inversion in the unipolar left arm lead, in vertical or semi-vertical hearts (Figure 26, V<sub>L</sub>b), is without adverse significance. Table 21 summarizes this situation. It is worthy of note that, of 445 cases having a vertical electrical axis, two-thirds showed upright T waves in Lead aVL (Figure 26, V<sub>L</sub>a).

TABLE 21  
T WAVE IN aVL DIPHASIC OR INVERTED  
WITH Q ABSENT AND S > R

163 Cases. Average Duration 2.8 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	52	0	2
Clinically Abnormal	5 (9%)	0	
<b>40 and over:</b>			
<b>65% of total</b>			
Clinically Normal	88	0	8
Clinically Abnormal	18 (17%)	2	

We then proceed to the various contours which are considered at least potentially dangerous.

We had exactly 100 cases showing diphasic or inverted T waves in aVL with a positively directed QRS less than 5 mm. in amplitude (Figure 26, V<sub>L</sub>c). Table 22 summarizes the experience with this pattern. Whereas 50 per cent of the cases in each age group with this aVL pattern showed associated cardiovascular abnormalities, the clinically abnormal controls totaled 19 per cent and 32 per cent respectively. Of the 42 normals, age 40 and over, 7, or 17 per cent, developed cardiovascular impairments, whereas in the control group only 9 per cent did so.

TABLE 22  
T WAVE IN aVL DIPHASIC OR INVERTED  
WITH QRS < 5 mm.

100 Cases. Average Duration 3.1 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	8	0	1
Clinically Abnormal	8 (50%)	0	
<b>40 and over:</b>			
<b>84% of total</b>			
Clinically Normal	42	0	7
Clinically Abnormal	42 (50%)	3	

With the T waves in aVL iso-electric or low, and the QRS mainly upright and exceeding 5 mm. (Figure 26, V<sub>L</sub>d), the picture is less favorable (Table 23). It will be observed that once more a high proportion were aged 40 and over, and that of these, 78 per cent had other abnormalities.

TABLE 23  
T WAVE IN aVL ISO-ELECTRIC OR LOW  
WITH QRS > 5 mm.

49 Cases. Average Duration 3.6 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	2	0	0
Clinically Abnormal	2 (50%)	0	
<b>40 and over:</b>			
<b>92% of total</b>			
Clinically Normal	10	0	1
Clinically Abnormal	35 (78%)	3	

Table 24 shows the group in which T waves in aVL were diphasic or inverted with the QRS exceeding 5 mm. in positive amplitude (Figure 26, V<sub>L</sub>e). It should be reiterated that all

T waves elsewhere in each tracing were normal. Once more the upper age group predominated heavily, as did the proportion of clinically abnormal cases. Very striking in this category are the numbers of normals 40 and above who developed abnormalities or died, and the high percentage of deaths among the abnormals. This can hardly be otherwise than significant, even in so small a group.

TABLE 24  
T WAVE IN aVL DIPHASIC OR INVERTED  
WITH QRS > 5 mm.

42 Cases. Average Duration 3.1 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	2	0	0
Clinically Abnormal	1 (33%)	0	
<b>40 and over:</b>			
<b>93% of total</b>			
Clinically Normal	10	1	3
Clinically Abnormal	29 (74%)	8	

The experience with diphasic or inverted T waves in Lead aVF, the QRS complexes being predominately negative (Figure 26, V<sub>F</sub>b), is shown in Table 25. Although the number of cases is small, comparison with parallel findings in vertical hearts (Table 21) suggests that this aVF pattern may not be simply indicative of a normal transverse heart.

A strikingly similar picture is presented in a much larger number of cases in which the T waves in Lead aVF were upright (Figure 26, V<sub>F</sub>a). Three-fourths of the cases with horizontal electrical axes showed positive T waves. It should be pointed out that this unipolar extremity QRS pattern will produce left axis deviation of the deep S2 type in the standard leads. The suspicion with which the latter has been regarded may thus be justified.

TABLE 25

T WAVE IN aVF DIPHASIC OR INVERTED  
WITH Q ABSENT AND S > R

29 Cases. Average Duration 4.0 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	6	0	3
Clinically Abnormal	3 (33%)	0	
<b>40 and over:</b>			
<b>69% of total</b>			
Clinically Normal	8	0	2
Clinically Abnormal	12 (60%)	1	

Table 26 shows the group with low upright QRS and diphasic or inverted T in Lead aVF (Figure 26, V<sub>F</sub>c).

TABLE 26

T WAVE IN aVF DIPHASIC OR INVERTED  
WITH QRS < 5 mm.

49 Cases. Average Duration 3.3 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	5	0	0
Clinically Abnormal	3 (38%)	0	
<b>40 and over:</b>			
<b>84% of total</b>			
Clinically Normal	20	0	2
Clinically Abnormal	21 (51%)	1	

Table 27 includes low upright and iso-electric T waves in Lead aVF, with high positive QRS complexes (Figure 26, V<sub>F</sub>d).

TABLE 27

T WAVE IN aVF ISO-ELECTRIC OR LOW  
WITH QRS > 5 mm.

15 Cases. Average Duration 3.9 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	3	0	0
Clinically Abnormal	5 (63%)	1	
<b>40 and over:</b>			
<b>47% of total</b>			
Clinically Normal	1	0	0
Clinically Abnormal	6 (86%)	2	

Finally, Table 28 deals with diphasic and inverted T waves in Lead aVF, again with high positive QRS complexes (Figure 26, V<sub>Fe</sub>).

TABLE 28

T WAVE IN aVF DIPHASIC OR INVERTED  
WITH QRS > 5 mm.

47 Cases. Average Duration 2.5 Years

	Number of Cases (% Clinically Abnormal)	Cardiovascular Deaths	Developed Other C-V Abnormalities
<b>Under Age 40</b>			
Clinically Normal	4	0	0
Clinically Abnormal	13 (76%)	0	
<b>40 and over:</b>			
<b>64% of total</b>			
Clinically Normal	12	0	3
Clinically Abnormal	18 (60%)	2	

The figures in connection with the aVF categories are not what we expected, in that we are unable to explain the seeming inconsistencies among the various groups. Nevertheless, pending the accumulation of more data, we still feel that these patterns deserve to be approached on a conservative underwriting basis.

Leaving out of consideration the vertical and horizontal electrical axis patterns, we must conclude from the material presented, which totalled 302 cases, that all of the categories are poor prognostically. Comparisons with the control group (Table 2) are unfavorable in every instance. It is important to point out that in numerous cases these T wave contours represent the only residua from proven infarctions. Certainly any case in which they occur should be subjected to the closest scrutiny. We do not feel that any of these patterns associated with abnormal clinical symptoms or signs are ordinarily compatible with insurability on any basis. The same conclusion applies to development of these contours in serial tracings which were initially normal.

#### *Conclusion*

Further summary would be nothing more than repetition. It is hoped that our analysis of these day-by-day problems will be helpful to others in formulating related underwriting practise.

**PRESIDENT YLVISAKER**—We are very much indebted to Dr. Kirkland and his associates for presenting such an excellent summary of their electrocardiographic studies and the relation of these studies to our underwriting problems. We hope others will contribute their experiences in the future and so add to our knowledge of electrocardiography in life insurance medicine.

Miss Annie Mary Lyle of The Prudential is one of the co-authors of the paper presented by Dr. Kirkland. We know her as a Fellow of The Actuarial Society who has become very interested in cardiovascular impairments and their relation to electrocardiography. She has contributed much to our underwriting knowledge in this field and I am glad to call on her to continue the discussion. Miss Lyle.

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Due acknowledgment is made to Dr. L. S. Ylvisaker, who initiated our whole electrocardiographic program and guided it through its first eleven years, and to Dr. P. V. Wells and Mr. Lionel Oliver, who contributed invaluable technical assistance.

MISS LYLE.—Actuaries have the reputation of taking no interest in a mortality study unless they can have thousands and thousands of cases. It is true, they cannot make a mortality investigation in the usual way unless they have a large amount of material. Being an actuary myself, I can sympathize with and understand their viewpoint.

But being Scotch by ancestry and Scotch by nature, as well as Scotch by profession, I have learned to make the most of what little I can scrape together. Fifty cases here, a hundred there—they make a picture, even if they do not make a mortality investigation. The small groups we have presented here today give us some guide for appraising risks, crude as it is, while we are waiting for the necessary volume to accumulate. Sometimes one case that tells us what we want to know is worth a thousand others. Several such cases are illustrated in the paper. It is well to remember that, aside from animal experiments, all we know about electrocardiography today has been learned from the study of small groups and single cases.

Moreover, it is my firm conviction that the careful study of small groups is the wisest kind of preparation for a large-scale mortality investigation. I think small groups are preferable to large ones for this purpose because they can be studied more closely. From this detailed study the significant characteristics of the impairment can often be determined and used in formulating the basic classification to be investigated. And it is the classification we use for the investigation that will, in the end, shape our rating schedule.

We have learned a great deal from the study of our Home Office employees. We have felt repaid over and over for the time and effort it has taken.

PRESIDENT YLVISAKER.—Dr. Paul Langner, Associate Medical Director of The Provident Mutual Life Insurance Company of Philadelphia, has also been very much interested in the application of electrocardiography in life insurance medicine. He has made previous contributions to our knowledge on this subject. I have requested him to add his comments. Dr. Langner.

DR. LANGNER — We have just heard a splendid paper. They have analyzed the subject and presented it in superb fashion. In fact, they have covered their subject so well that there is little for anyone to add.

When applicants for insurance have isolated electrocardiographic abnormalities such as the authors have discussed, they present quite a problem in underwriting. The authors' analysis of their results will certainly help to guide us in our thinking. In these borderline problem cases, what else can we do to help evaluate the risk? We think it is helpful to employ three additional procedures. They are as follows:

1. In borderline T wave changes, we interpret the electrocardiogram by vector methods. This can be performed very easily and quickly on routine demonstrations obtained from the field which consist of the three standard limb leads and the six precordial V leads. If the angle formed by the mean vector of the QRS and T is normal, we consider the T waves as being within normal limits.
2. The second procedure which we use is the electrocardiogram after exercise. This is done by following the Master technic, using the double test.
3. The third procedure which we have been using recently is the ballistocardiogram. Its exact place in underwriting has not yet been definitely determined, but it promises to be very useful, especially in the age group under fifty. The ballistocardiogram gives valuable information about the heart that is totally different from the electrocardiogram. In fact, in a given case the electrocardiogram has been perfectly normal and the ballistocardiogram quite abnormal, and vice versa. We think it is going to be very helpful to use both tests.

I have just one more comment. In the past two years we have had quite a change of opinion about incomplete right bundle branch block. We agree heartily with the authors that it is of no consequence and may be disregarded in the absence of other impairments.

On the other hand, complete right bundle branch block must be regarded with some reservation. We believe that if it occurs in the age group from twenty to thirty-five years, or if it is known to have been present for ten years or more without change—in the absence of any history, with normal clinical examination, and with normal tests of heart function such as I have mentioned—it must not be taken too seriously.

What I have said for right bundle branch block I think applies more or less to most of the borderline electrocardiographic abnormalities.

PRESIDENT YLVISAKER—Thank you, Dr. Langner.

Calcification of the aorta as seen in chest x-ray films submitted for review in connection with our underwriting has, during the last twenty years, given us repeated problems. The significance of this finding, from a prognostic standpoint, has caused much discussion. The New York Life has reviewed their experiences and Dr. Murray F. Bell will present the result of their studies. Dr. Bell.

## CALCIFICATION OF THE THORACIC AORTA: A MORTALITY STUDY

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A calcific aortic plaque identified radiologically constitutes reliable evidence of atherosclerosis (1, 2). Aschoff (3) described the pathologic development of these intimal lesions of atherosclerosis and observed their predilection for the lower abdominal and posterior thoracic aortic areas. Roentgenologically, a calcific plaque is most frequently demonstrated in the lower abdominal aorta near its bifurcation. In the thoracic aorta, it is most often seen in the aortic knob. Its occurrence in the ascending aorta supports a diagnosis of luetic aortitis (4). Although such calcification, too, is believed to be a manifestation of atherosclerosis accelerated by syphilitic infection. Degenerative medial changes bring about a loss of elasticity producing elongation and dilatation of the aorta which usually, but not invariably, accompany the intimal sclerosis. Blumenthal et al (5) described the frequent occurrence of calcium deposits in the media of the human aorta, as demonstrated by special stains and microincineration. However, the relatively homogenous concretions, demonstrable roentgenologically in the aorta, are generally conceded to be of intimal origin. This should not be confused with the diffusely distributed medial calcification of the peripheral muscular arteries, where an entirely different process prevails, the so-called Monckeberg's sclerosis.

Goodman et al (2), in studying a series of diabetics, found a high incidence of calcific aortic plaques in conventional

chest films (41.5%). They stated in 1950, "The finding of calcified plaques in the aortic knob in the chest film is a valuable sign of atherosclerosis, and certainly there is no better means of demonstrating atherosclerosis in its advanced stages. The rather high incidence of this finding is striking, although sufficient emphasis has not been given to it in the current literature." They did not comment on the incidence or degree of any accompanying aortic elongation or dilatation. Furthermore, they consider that demonstrable atherosclerosis in the upper aorta is generally a good index of the process in its tributaries, i. e., the coronary and cerebral arteries. Willius et al (6), studying 5,060 consecutive postmortem examinations, concluded that fairly close parallelism occurred between the degrees of coronary and aortic sclerosis; but a study of individual components revealed much discrepancy in many instances.

This investigation was undertaken to study the prognostic import of a calcific plaque in the thoracic aorta.

#### *Material and Methods*

The cases were taken from the files of the Medical Department of the New York Life Insurance Company. A review of x-rays taken between 1931 and 1946 yielded 182 cases demonstrating a calcific plaque in the thoracic aorta. The calcifications were all situated in the aortic knob, none having been observed in the ascending aorta. Of these 182 cases, 127 were insurance applicants and 55 were disability claimants. A history, physical examination, electrocardiogram and teleoroentgenogram had been completed in all cases at sometime between 1931 and 1946. The majority was examined at the Home Office where the chest x-rays were taken in the posteroanterior position, at a target distance of 6 feet, with an exposure of 1/20 second. Chest films presenting plaque-like shadows which could not be confidently differentiated from densities produced by rib markings, transverse processes of vertebrae, or bronchial calcifications, had been discarded. Although such cases could undoubtedly have been clarified by utilizing special x-ray technique (1, 7,

8, 9) such as overpenetration, stereoscopy and tomography, the individuals were, unfortunately, unavailable for further study. All cases presenting more than slight elongation or dilatation of the aorta, cardiac enlargement, abnormal cardiac contour, electrocardiographic abnormalities, significant lung pathology, or chest deformity were eliminated from the statistical study to be described, mainly because of the lack of homogeneity of this material. Finally, those presenting pertinent history, physical findings, or laboratory data relative to cardiovascular or other significant systemic disease, were also deleted for the same reason, with exceptions as noted below. From the original total of 182 cases, only 33 remained which fulfilled the criteria established for this study. Of these 33 cases, all insurance applicants, 21 would have been considered standard risks were it not for the discovery of the aortic plaque. A few of these had a history or finding of some additional minor impairment, i. e., mild psychoneurosis, slight to moderate asymptomatic thoracic scoliosis, trigeminal neuralgia, otosclerosis, and old healed minimal apical scarring. These findings would not have prevented the issuance of standard insurance. The remaining 12 cases presented an additional finding which would have necessitated a slightly increased premium, based on an estimated mortality of 125 per cent to 150 per cent of standard. In almost all of these cases, this additional finding was either a slight systolic blood pressure elevation recorded on multiple observations as 150 to 155 mm. Hg. with normal diastolic levels, or a systolic murmur described as apical and/or basal, grade II, non-radiating, unassociated with cardiac enlargement. Since sclerosis of the aorta frequently produces a systolic blood pressure elevation and/or a systolic murmur, it is questionable whether these findings, present in slight degree in some of our cases, should warrant a separate rating, or whether the one rating for the atherosclerotic aortic process should suffice for both findings. These individuals were retained in the study, inasmuch as we were reluctant to discard cases which, aside from the aortic plaque finding, were only slightly below average. In addition, we considered it of interest, notwithstanding the

small number of cases involved, to determine whether the mortality of the slightly substandard group was in excess of that found in the standard group.

For statistical analysis the 33 cases were divided into two groups. Group I consisted of the 21 standard risks, and Group II consisted of the 12 slightly substandard risks as described above. The age incidence at the time of examination ranged between 40 4/12 and 70 years for the entire series, with an average of 53½ years in Group I and 52 years in Group II. Since individuals beyond the age of 65 years are not accepted for new insurance by this company, the number of cases beyond the sixth decade was limited by this factor. Men predominated, there being only two women, one in each group. In view of the fact that some of the individuals were followed from 1931 and others from 1946, allowance was made for the differences in length of exposure. The procedure of tracing the cases was begun in the latter part of 1950 and completed early in 1951. All cases were traced. Information relative to the dates and causes of death was obtained from various sources such as death certificates, attending physicians' reports and hospital records. Deaths resulting primarily from cardiovascular diseases were segregated from those attributed to other causes. The number of deaths from all causes was compared with the expected number of deaths. Similarly, the number of deaths resulting from cardiovascular disease was compared with this corresponding number of deaths. The expected mortality was based on company actuarial tables for standard issues, covering approximately the same period of exposure as the cases in the study.

#### *Results and Discussion*

There were 10 deaths in this series of 33 cases. These are listed in Table A. In Group I, 6 deaths occurred, 3 being due to cardiovascular disease. In Group II, there were 4 deaths, 2 being due to cardiovascular disease. The results of the statis-

TABLE A  
DEATHS FROM CARDIOVASCULAR CAUSES

		<u>Age at Exam. (Yrs.)</u>	<u>Age at Death (Yrs.)</u>
Group I	1. Arteriosclerotic Heart Disease (Diabetes Mellitus)	44 1/12	51
	2. Coronary Occlusion	70 1/12	70 4/12
	3. Cerebral Hemorrhage (Bronchopneumonia)	62 1/12	74 7/12
Group II	4. Cardiac Decompensation	51 1/12	64 4/12
	5. Coronary Occlusion	57	57 11/12
DEATHS FROM OTHER CAUSES			
Group I	1. Carcinoma Bladder	63 6/12	71 6/12
	2. Brain Tumor	64 9/12	71 1/12
Group II	3. Carcinoma Lung	50 9/12	52 8/12
	4. Pneumonia	42 9/12	45 9/12
	5. Gangrenous Appendicitis	57 3/12	61 11/12

tical analysis are presented in Table B. In Group I, the ratio of actual to expected deaths from all causes is 162 per cent, and from cardiovascular causes, 163 per cent. In Group II,

TABLE B  
Ratios of Actual to Expected Deaths  
for Lives with Calcific Plaque of the Thoracic Aorta  
(Expected Deaths Based on Company Experience under Standard Issues.)

	Actual Deaths	Expected Deaths	Ratio of Actual to Expected Deaths	
			Group I — 21 Lives	
(Cases where additional impairments were rated not more than plus 25)				
Deaths from All Causes	6	3.70		162%
Cardiovascular Deaths	3	1.84		163%
(Cases where additional impairments were rated between plus 25 and plus 50)				
Deaths from All Causes	4	1.19		336%
Cardiovascular Deaths	2	.60		333%

the ratio of actual to expected deaths from all causes is 336 per cent and from cardiovascular causes, 333 per cent. If the

expected number of deaths in Group II were increased by 50 per cent to reflect the approximate effect of the additional impairment, the ratios of actual to expected deaths would be 223 per cent for deaths from all causes, and 222 per cent for deaths from cardiovascular causes.

The mortality ratios in the preceding table suggest the following tentative hypotheses:

- (a) The mortality rates that will be experienced on lives with thoracic aortic plaques and no other significant impairment might be somewhat higher than the mortality rates on standard lives but not alarmingly so.
- (b) The mortality rates that will be experienced on lives with thoracic aortic plaques accompanied by even a relatively minor additional cardiovascular impairment might be considerably higher than the mortality rates on standard lives. Furthermore, the sum of the two ratings will not generally cover the hazard in these cases, suggesting that the additional finding reflects a more advanced pathologic process. This statistical finding with respect to multiple impairments is in accord with the observations of Bolt (10) and Evers (11) in their independent studies.

Again it must be stressed that from a total of 182 cases demonstrating calcification in the knob of the aorta, only 21 could be found in which this abnormality appeared as an isolated finding. The vast majority of the remaining 161 cases was deleted from the statistical study because of associated cardiovascular abnormalities such as significant history, electrocardiographic changes, arterial hypertension, cardiac enlargement, organic murmurs, significant elongation or dilatation of the aorta, cardiac decompensation, or evidence of cerebral vascular disease. This appears to agree with the concept that atherosclerosis of the thoracic aorta is generally associated with similar involvement of its coronary and cerebral tributaries or with other significant cardiovascular abnormalities. However, as regards those relatively infrequent cases in which the plaque appears as an isolated abnormality, in the light of our limited statistical

study the prognostic import relative to longevity is not grave, suggesting a more localized, less progressive process in these individuals.

In conclusion, we wish to emphasize that the number of cases in this study is not large enough to permit precise statistical validity to be attached to the above hypotheses. They are considered to be tentative, and are being presented with the aim of inducing others to offer additional experience.

#### *Summary*

One hundred eighty-two cases presenting radiologic evidence of a calcific plaque in the aortic knob were reviewed, and the following observations made:

1. A large majority, 152 individuals, presented additional cardiovascular abnormalities.
2. In 21 individuals a calcific plaque appeared as an isolated abnormality. A mortality study of this group revealed some increase in the cardiovascular death rate, but not to any great extent.
3. Twelve individuals presenting relatively minor additional cardiovascular impairments were also statistically studied. A considerably higher cardiovascular mortality rate was sustained by this group.

Conclusions drawn from the above observations are considered to be tentative because of the small number of cases upon which the statistical analysis was based.

#### *Addendum*

Since the completion of this study, additional material was made available to us through the cooperation of Dr. H. H. Fellows, Associate Medical Director, Metropolitan Life Insurance Company. One hundred and eight employees of that company showed calcific aortic plaques in chest roentgenograms taken during the years 1930 to 1950. The plaques appeared as isolated abnormalities in 63 individuals, of

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The authors are indebted to Mr. Charles M. Sternhell, Executive Assistant, Underwriting Research Division, and his associates for their analysis of the statistical data.

whom 30 were men and 33 women. Statistical analysis of the male employees, based on the same table used in the original study, revealed a mortality ratio of 108 per cent. The mortality experience of the women was even more favorable. It is possible that the difference between the mortality rate of 108 per cent and the corresponding one of 162 per cent obtained in the original study may be entirely due to statistical fluctuation. It could also be caused by differences in the type of material as follows:

1. Information relative to the absence of pertinent clinical history is more likely to be reliable when elicited from employees.
2. Serial films, available in the employee group, afforded the opportunity of confirming previous questionable findings with the attendant probability that plaques were observed at an earlier stage of their development. Consequently, an increased longevity would be expected in such cases. This may also explain the considerably higher incidence of uncomplicated cases in this entire series as compared to ours, even after elimination of the disability cases which constituted a part of our material.

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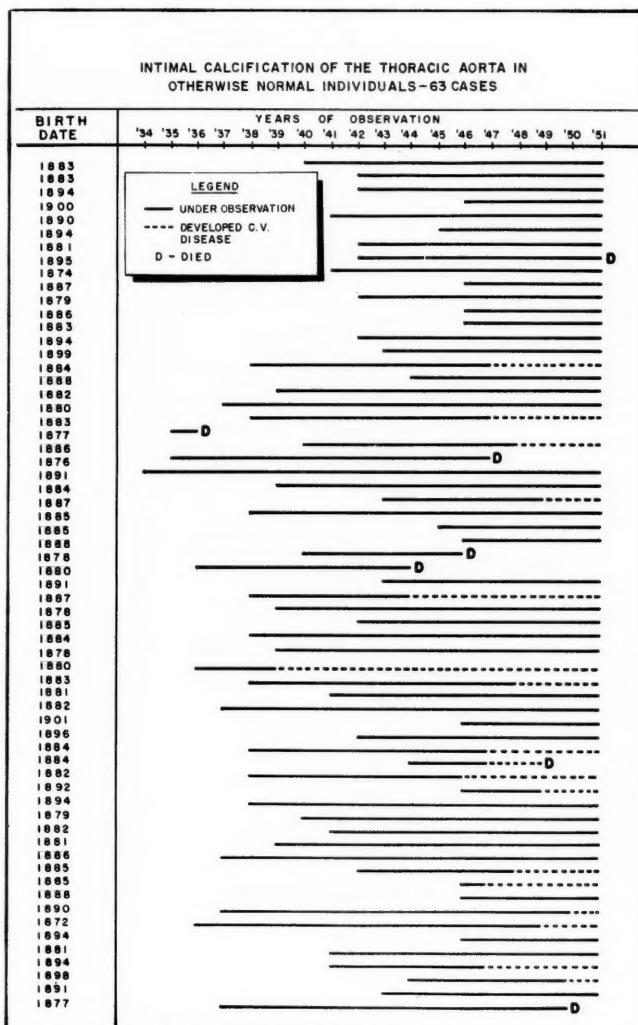
**PRESIDENT YLVISAKER**—The Prudential has also reviewed their cases showing this abnormality, and Dr. Kiessling will present a summary of their experience.

**DR. CHARLES E. KIESSLING**—I should like to compliment Drs. Bolt and Bell for their timely and interesting presentation. Inasmuch as many of us have not had a large experience with aortic intimal calcification, and because we do not know how to evaluate this lesion, I think we should welcome any discussion on the subject, even though the group presented is not very large. We all know that many of our ideas of prognosis in clinical medicine are based upon relatively small groups. However, we must be cautious in evaluating a lesion which may have a late increased mortality, and, as you are well aware, we must be careful in dealing with applicants for insurance who do not give us a complete history.

Dr. Phillip V. Wells and I have been assembling cases of aortic intimal calcification for several years. However, when we discard all those cases showing associated cardiovascular pathology—a large part of them—we end up with comparatively few cases. It is this high incidence of associated cardiovascular pathology that is one of the main reasons why we should hesitate to offer standard insurance to applicants for insurance showing aortic calcification.

From among our employees we have collected 48 cases of aortic intimal calcification in persons otherwise normal, and 15 similar cases from the insurance selection file. Each individual included in the group of 63 cases was first observed between the years of 1934 and 1946. Cases discovered subsequent to 1946 were not included. The study was closed

CALCIFICATION, THORACIC AORTA: 145



in September, 1951. The average duration of observation was 10 years. In the chart, the left-hand column gives the birth date of each individual presented. The beginning of the black line indicates when the case was first discovered. At this time the individual was otherwise normal; he had no hypertension or history of hypertension; he had no detectable heart murmurs; none of them had diabetes, and all had normal electrocardiograms. Except for the first 15 cases, insurance applicants about whom we could not be sure, none had any history of chest pain. Where the line becomes broken, that individual developed definite evidence of cardiovascular disease in the form of hypertension, abnormal electrocardiogram, or, in a few of the cases, typical angina. Those who died were so designated with this "D". Slightly more than half of them were cardiovascular deaths. The women in the group, of whom there were 18, did much better than the men.

On such a small group I do not like to mention mortality figures, but I think I should say that the mortality was slightly better than normal, and very close to normal when the 18 women were excluded. The mortality estimate was based upon the basic table of 1946-1949.

I think this group and the group from the Metropolitan give a fairly accurate estimate and portrayal of what happens to people with aortic intimal calcification who are otherwise absolutely normal as far as can be determined from the means we have available for examining them.

There is one other fact which I would like to bring to your attention. When I speak of aortic intimal calcification, I am not speaking of atherosclerosis as contrasted with the absence of atherosclerosis. In this study, we have singled out for investigation those individuals who have a deposit of calcium in their atherosclerotic lesion localized in a certain small part of the aorta, the knob, where it can be visualized on a routine chest x-ray. I need not call to your attention the fact that an underexposed x-ray, where the aortic and cardiac shadows are practically white, will not reveal a small calcific plaque even though it may be present.

## CALCIFICATION, THORACIC AORTA: 147

I think the whole situation can be summarized about as follows: If an elderly woman shows an arteriosclerotic plaque and is otherwise normal, the finding is probably without significance; if a man has an arteriosclerotic plaque and is otherwise normal, the significance is slight, if the finding has any significance at all. In other words, if an applicant for insurance shows a calcific plaque, I think he can be insured, but perhaps with a small rating, just to play safe, because of the high incidence of associated cardiovascular pathology in individuals who show this lesion.

It will be interesting to see how time and further investigations deal with the opinions expressed here this afternoon.

**PRESIDENT YLVISAKER**—We are indebted to Dr. Bell and Dr. Bolt for their presentation. I also want to thank Dr. Fellows for his contribution of the Metropolitan material and Dr. Kiessling of the Prudential for giving us their experience to date. They have given us a basis for future consideration of this impairment and we hope others will contribute more information on this subject in the future.

Dr. James R. Gudger of The Mutual Life of New York has been teaching insurance medicine at The New York University Post-Graduate Medical School and happened to show me an outline he had prepared on cardiovascular impairments for his students. I felt that this outline would be valuable for all of us in our discussion of these impairments with our medical examiners. I have asked him to present this outline and make it available for us in our Transactions. Dr. Gudger.

**DR. JAMES R. GUDGER**—Mr. Chairman, Members of the Association: I shall take only a very brief moment to describe the main features in the form and content of this paper being published in the next issue of the Association's Transactions under the title of "The Life Insurance Examiner and the Cardiovascular System."

The paper, as mentioned by Dr. Ylvisaker, is only an outline of a talk, the subject of which was, "The Heart and the Life Insurance Examiner," given before post-graduate

students in cardiology at the University Hospital. It is being made available to the members of the Association for possible use in their activities, either educational activities or talks to examiners or other groups of the medical profession who may be interested in this phase of life insurance medicine and who are not familiar with the Home Office procedures of risk selection.

It is not being presented in detail because, of course, it is only in outline form. In fact, there are no new scientific data included, nor any new information which is not known to the membership here, or easily available to them. It is simply a collection and arrangement of the data which might interest groups, as I have mentioned. It concerns, chiefly, the requirements needed at the Home Office to underwrite impairments of the circulatory system. In this talk we try to tell about the things we want from examiners, how we want them reported, and what we do with them when they are received at the Home Office. It also includes some fundamentals of the numerical method of risk selection and rating. Also, it includes some of the mortality statistics and experience to be expected among the principal impairments of the cardiovascular system.

I should be glad to furnish a copy of this outline to anyone interested in using it before it appears in the next volume of the Transactions early in the coming year.

THE LIFE INSURANCE EXAMINER  
AND  
THE CARDIOVASCULAR SYSTEM\*

James R. Gudger, M. D.  
*Medical Director*

*The Mutual Life Insurance Company of New York  
New York City*

*Introduction*

I. *General Considerations*

A. Professional Relationship

1. To Life Insurance Companies
  - a. As *Examiner*—examinations—usual history and physical examination, applicants for new insurance
  - b. As *Consultant*—cardiovascular survey (*short*—consisting of EKG and x-ray or orthodiagram, *long*—in more detail for disability or complicated diagnostic cases)
  - c. As *Expert Witness*—for either side in litigation of death or disability claims
  - d. As *Attending Physician*—statements to Company form permanent records—contents carefully protected and confidential
2. To Agents
  - a. Opinion of Company not to be based on agent contacts
  - b. Examination results not to be given to agent
  - c. Medical opinion not to be influenced by agent

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\*Outline of a lecture before class in Cardiology at New York University-Bellevue Medical Center, Post-Graduate Medical School, May 11, 1950 (Revised August 1951)

- B. Reports—to include
  - 1. Findings, detail
  - 2. Opinion, clearly as possible and basis for
  - 3. Qualifications, state yours as facts to help those concerned

## II. *Mortality*

- A. Experience based on combined records of many companies, many thousands of cases in major impairments
- B. "Mortality ratio", or per cent of mortality, defined as ratio of ACTUAL to EXPECTED mortality
  - e.g.—in a group 10 die, 10 expected,  $M=100\%$
  - but—in a group 20 die, 10 expected,  $M=200\%$

### Effect on premium rates:

Standard rates up to 125 per cent of normal mortality  
—if higher usually charge extra premiums.

- C. Mortality in life insurance based on selected groups
- D. Longevity predicted on average person in general population by death rate statistics
- E. Build (height and weight) e.g. 6 feet, 228 pounds= $M$ , 140%
  - 1. American Experience Table (obsolete)
  - 2. Commissioners 1941 Standard Ordinary Mortality Table—(generally lower mortality rates in younger age groups as compared to American Experience Table) Figure 1

COMMISSIONERS 1941 STANDARD ORDINARY MORTALITY TABLE

Age	Number Living	Number Dying	Death Rate per 1000	Average Future Lifetime Years
0	10,231.0	231.0	22.58	62.33
1	10,000.0	57.7	5.77	62.76
2	9,769.3	30.2	4.14	62.20
3	9,901.1	33.4	3.38	61.37
4	9,867.7	29.5	2.99	60.58
5	9,838.2	27.2	2.76	59.76
6	9,811.0	25.6	2.61	58.92
7	9,783.4	24.1	2.47	58.08
8	9,761.2	22.5	2.31	57.22
9	9,738.7	20.7	2.12	56.35
10	9,718.0	19.1	1.97	55.47
11	9,695.3	18.6	1.91	54.59
12	9,680.4	18.6	1.92	53.68
13	9,661.8	19.1	1.98	52.78
14	9,642.7	20.0	2.07	51.89
15	9,622.7	20.7	2.15	50.99
16	9,603.7	21.6	2.23	50.09
17	9,581.0	21.6	2.25	49.21
18	9,559.4	22.0	2.30	48.32
19	9,537.4	22.6	2.37	47.43
20	9,514.8	23.1	2.43	46.54
21	9,491.7	23.1	2.51	45.65
22	9,467.9	24.5	2.59	44.77
23	9,443.4	25.3	2.68	43.88
24	9,416.1	26.1	2.77	43.00
25	9,382.0	27.1	2.88	42.12
26	9,346.8	28.0	2.99	41.24
27	9,336.9	29.0	3.11	40.36
28	9,307.9	30.0	3.25	39.49
29	9,276.6	31.5	3.40	38.61
30	9,246.1	32.9	3.56	37.74
31	9,213.2	34.4	3.73	36.88
32	9,178.8	36.0	3.92	36.01
33	9,141.8	37.9	4.12	35.13
34	9,105.2	39.9	4.35	34.29
35	9,065.5	41.6	4.59	33.44
36	9,023.9	43.8	4.86	32.59
37	8,980.1	46.3	5.15	31.75
38	8,938.8	48.0	5.46	30.91
39	8,885.0	51.6	5.81	30.08
40	8,833.4	54.6	6.18	29.25
41	8,778.8	57.8	6.59	28.43
42	8,720.0	61.3	7.03	27.62
43	8,659.7	65.1	7.51	26.81
44	8,594.6	69.1	8.04	26.01
45	8,525.5	73.4	8.61	25.21
46	8,452.1	78.0	9.23	24.43
47	8,374.1	83.1	9.91	23.65
48	8,291.1	88.2	10.64	22.88
49	8,202.9	93.9	11.45	22.12

\*Average Future Lifetime is sometimes called "Expectation of Life."

COMMISSIONERS 1941 STANDARD ORDINARY MORTALITY TABLE

Age	Number Living	Number Dying	Death Rate per 1000	Average Future Lifetime Years
50	8,109.0	99.9	12.32	21.37
51	8,009.1	106.3	13.27	20.54
52	7,899.3	113.7	14.20	19.71
53	7,789.8	120.2	15.43	19.19
54	7,669.6	127.7	16.65	18.48
55	7,541.9	135.6	17.98	17.78
56	7,413.3	143.9	19.33	17.40
57	7,282.4	152.4	21.00	16.43
58	7,109.9	161.5	22.71	15.77
59	6,948.4	170.7	24.57	15.13
60	6,777.7	180.2	26.59	14.50
61	6,635.7	187.5	28.37	13.86
62	6,407.6	199.8	31.18	13.27
63	6,207.8	209.6	33.76	12.69
64	5,996.2	219.4	36.58	12.11
65	5,778.8	229.0	38.40	11.55
66	5,558.8	238.7	42.96	11.01
67	5,311.3	247.3	46.56	10.48
68	5,064.0	256.0	50.40	9.97
69	4,805.5	263.0	54.00	9.47
70	4,545.5	269.6	59.30	8.99
71	4,275.9	274.8	64.27	8.52
72	4,001.1	278.7	69.66	8.08
73	3,724.4	284.0	75.00	7.72
74	3,441.4	281.6	81.81	7.23
75	3,159.8	280.1	88.64	6.82
76	2,879.7	276.5	96.02	6.44
77	2,602.2	270.7	103.99	6.07
78	2,332.5	265.0	112.59	5.72
79	2,069.9	252.2	121.86	5.38
80	1,817.7	239.7	131.85	5.06
81	1,578.0	225.0	142.60	4.75
82	1,332.0	206.6	151.16	4.46
83	1,144.4	190.6	168.57	4.18
84	953.8	171.6	179.88	3.91
85	782.2	151.8	194.13	3.66
86	630.4	132.0	208.37	3.42
87	494.4	113.8	225.33	3.19
88	385.9	93.7	243.00	2.98
89	292.2	76.4	261.44	2.77
90	215.8	60.7	280.99	2.58
91	161.1	46.8	301.73	2.39
92	108.3	30.9	323.64	2.21
93	73.3	25.4	346.66	2.03
94	47.9	17.6	371.00	1.84
95	30.1	11.9	396.21	1.63
96	16.2	5.6	427.19	1.37
97	10.1	3.6	548.26	1.08
98	4.5	3.2	724.67	.78
99	1.3	1.3	1,000.00	.50

\*Average Future Lifetime is sometimes called "Expectation of Life."

Figure 1.

## F. Selection

## 1. Dangers

- Samples too small to indicate true mortality
- Individual vs. group (e.g. *some* persons with double mitral-aortic murmurs *may* live long)

## 2. Basis—Figure 2

- build
- family history
- medical history
- physical impairments
- habits
- occupation
- social status and environment
- others

} medical

Figure 2

Items used as basis of selection in life insurance.

## 3. Rating

a. Work sheet—sample—Figure 3

Girth Credit	Plan Credit	W.P.	D.I.	G	P	W.P.	D.I.
lbs. Build							
Family History							
Blood Pressure							
Medical History							
Physical Condition							
Others							
<b>MEDICAL RATING (Tot.)</b>							
Plan							
Occupation							
Habits							
<b>TOTAL RATING</b>							
<b>Medical Director Recommendations</b>							

Figure 3.

Numerical rating work sheet.

*Cardiovascular System—Partial List of More Important Conditions*

## I. Impairments

## A. Murmurs

## 1. Difficulty in detection

- a. All characteristic signs not demonstrable
- b. Etiology of some never determined

## 2. Examination technic—Figure 4

NAME OF INSURED (Please Print)		DATE OF BIRTH		
SIGNATURE OF INSURED (For Identification)				
PLEASE ENCIRCLE ALL TERMS WHICH APPLY				
1. IS THERE A HEART MURMUR?		Yes	No	
(a) TIME:		Systolic	Presystolic	
(b) AREA:		Aortic	Pulmonic	
(c) INTENSITY:		Faint	Loud	
(d) TRANSMISSION:		Yes	No	
Direction:				
2. IS MURMUR CONSTANTLY PRESENT?				
(a) Before AND after exercise		Yes	No	
(b) Through BOTH inspiration and expiration		Yes	No	
(c) In BOTH erect and recumbent positions		Yes	No	
3. IS HEART ENLARGED?		Yes	No	
Slight		Moderate	Marked	
4. IS THERE ANY				
Edema?		Cyanosis?	Dyspnea?	
5. IS THERE ANY HISTORY OF				
Rheumatism? Chorea? Severe Infection?				
(Give dates and full details or state "None")				
6. IS THE PULSE				
Regular? Irregular? Intermittent?				
7. EXERCISE TEST (Describe the type and amount of exercise)				
(a) IRREGULARITIES PER MIN.		Before Exercise	Immediately after Exercise	
				2 Min. after Exercise
(b) PULSE RATE				
(c) BLOOD PRESSURE				
8. WHAT IS YOUR DIAGNOSIS AND OPINION?				
Date _____		Medical Examiner _____ M. D.		

Figure 4.

Example of a "Heart Chart"

- Place—quiet, facilities lie down, adequate light
- Position—upright, recumbent
- Special conditions
  - Before and after exercise (use of Bowles stethoscope recommended)
  - Left side (mitral murmurs)
  - Sitting, lean forward (aortic murmurs)

### 3. Classification

#### a. Essential Features—Figure 5

#### HEART MURMURS AND ABNORMAL HEART SOUNDS

**CLASS 2**

Also called functional by examiner:

<b>Systolic apical or precordial, not transmitted</b>	<b>most favorable</b>	<b>less favorable</b>	<b>least favorable</b>
<b>Systolic aortic or basic</b>	<b>=</b>		
<b>Systolic tricuspid</b>			

### CLASS 3

Called organic by examiner or with characteristics of organic or congenital murmur:

Pulmonic systolic widely transmitted (comp.)	
Mitral regurgitation; apical or precordial systolic transmitted to left	
Mitral stenosis; apical or precordial diastolic or presystolic	least favorable
Mitral regurgitation and stenosis	
Aortic stenosis; rough aortic systolic transmitted up with weak or absent A2	
Aortic stenosis suspected (not characteristic)	
Aortic regurgitation; soft diastolic blow from base downward along sternum	
Aortic stenosis and regurgitation	

Treat as P

## A method of classifying heart murmurs and abnormal heart sounds for life insurance purposes

- 1) Phase cardiac cycle
- 2) Point maximum intensity
- 3) Effect special conditions
- 4) "Transmission"

b. Diastolic

- 1) Nearly all organic
- 2) Estimated 5 per cent of all murmurs reported by examiners diastolic, e.g. mitral stenosis

- 3) Mortality 450 to 500 per cent
- c. Systolic—classic description best clinical guide, Levine (1)
  - 1) *Functional*
    - a) Most frequently heard, systolic, constant or variable, pulmonic area, ages under 40
    - b) Also rare musical murmurs, heard anywhere
    - c) Usually standard risks
  - 2) *Questionable Functional*
    - a) At *apex*, constant and inconstant systolic, 19 per cent of experience in one company (often organic in persons over 40)
    - b) Along left sternal border
    - c) Most *other* systolic murmurs considered organic—e.g. *mitral regurgitation* or *insufficiency* with history rheumatic fever within five years—300 per cent mortality
    - d) Etiology and significance some murmurs questionable—describe these carefully
  4. Reference and guide for clinical examination of heart without help of any instrument other than the stethoscope—pamphlet, "Examination of the Heart", American Heart Assn., 1775 Broadway, New York 19, N. Y. (1951)

B. Blood Pressure Variations

  1. Requirements
    - a. Use average of two readings on separate days
    - b. Seated, relaxed
    - c. Diastolic at 5th phase
    - d. Always in afternoon or evening if hypertension suspected, or history of

2. Normal—average for age in healthy persons (2) e.g. mean blood pressure age 50-54 (male) 134/83
3. Labile blood pressure
  - a. Criteria—e.g. 150/90 dropping to 136/80 clinical studies (few)
  - b. References
    - 1) Levy et al (3) (4) fluctuating blood pressure and transient elevations (150/90) studies and followup on 10,000 army officers at age 25 years—*results higher mortality rate and higher disability rate*. Disability retirements by age 60 estimated 69 per cent higher in this group, death rate from cardiovascular renal disease doubled.
    - 2) Diehl and Hesdorffer (5) material 153 students with systolic over 140 and systolic under 140. Ten-year followup, 22 per cent group with blood pressure 140 or over *had hypertension*, as against 4.5 per cent of those with systolic under 140.
    - 3) Insurance company records of blood pressure mortality now on about *1/4 million deceased persons* (6). Invariably mortality higher among persons with so-called labile blood pressure. Estimated that 15 per cent of persons examined fall in that classification

#### C. Arteriosclerosis and Coronary Disease

##### 1. Problem

- a. Early detection
- b. Careful history
- c. Early signs
  - 1) Peripheral arteries or retinal vessels
  - 2) Cardiac tone
  - 3) Response to exercise
  - 4) Color

## 2. Analysis with Dr. Willis (7)

a. Source of material—600 living policyholders vs. 600 deceased within 5 years of issue, cause given as coronary disease, similar groups in age, sex, amount, etc. Average age issue 49, death 52 years.

## b. Possibly significant factors—

- 1) *Overweight*—60 per cent of dead above optimum weight
- 2) *Family history*—51 per cent cardiovascular renal disease in immediate family in dead group, 43 per cent in living
- 3) *Occupation*—33 per cent more “professional” types in dead group

## D. Pulse aberrations

1. Tachycardia—usually acceptable in younger age groups (40 and below) up to 94 per minute
2. Bradycardia—questionable risk if below 55 per minute
3. Other arrhythmias—graded according to apparent significance, severity, and associated factors (EKG essential)

## E. Effort Syndrome (neurocirculatory asthenia)

1. Surprisingly few seen applying for insurance (estimated by some at 10 per cent among private patients)
2. Problem—differentiate from organic disease
  - a. Very careful history
  - b. Negative organic findings in C-V system
  - c. Usually accepted on basis of psychoneurosis if uncomplicated by other organic disease

## F. Other Conditions

1. Pericarditis—accept after recovery from acute type, mortality rates high on others
2. Acute rheumatic fever—more favorably regarded after about 5 years if not complicated

3. Rare conditions (such as traumatic lesions) considered on degree structural impairment and residuals
4. Congenital heart disease—mostly uninsurable, more favorable after surgical cure

## II. *Special Diagnostic Studies*

### A. Electrocardiogram

1. Required in
  - a. Suspected myocardial or conduction system lesions
  - b. Arrhythmias
  - c. Amounts
  - d. Ages
2. By
  - a. Cardiologist
  - b. Consultants
  - c. Company Examiners
3. Usually leads 1, 2, 3 also V, CF, or CR, 2, 4, and 5
4. Mortality
  - a. Normal—generally abnormalities without clinical significance cardiovascular disease, e.g. Kent bundle branch conduction defect; small number premature contractions; deep  $Q_3$
  - b. Less favorable—changes of clinical significance indicative of organic or possible organic disease, e.g. T wave changes; auricular fibrillation (or recent history of it); severe grades of block

### B. X-ray and Orthodiagram

1. Requirements
  - a. Usually single film, teleoroentgenogram of good quality
    - 1) Read from Clark-Ungerleider table (average Transverse Diameters of heart and aorta based on height and weight) 1938, 1640 cases, Mutual Benefit-Equitable Life Assurance Society, Am. Heart J. 24:494, Oct. 1942

## 2. Mortality

- a. Normal—changes in size or contour of chambers, or aorta, within 10 per cent of average
- b. Less favorable—marked degree above variations

III. *Disability Examinations*

## A. Many Cardiac

## 1. Requirements

- a. Opinion as to ability to work detailed and well supported
  - 1) Based on
    - a) Examiner's opinion (usually cardiologist)
    - b) Evidence from other investigation, anoxemia test not often used (danger)
    - c) Complete physical examination always advisable
    - d) *Hospitalization for investigation and study*

2. Most desirable way to report cardiac examinations—by criteria New York Heart Association, i.e. Etiologic, Anatomic, Physiologic, and Functional diagnosis

B. Notorious case, convictions in New York 1938, based on coaching insured people, digitalis poisoning, changes in electrocardiogram

(Dr. F. O. Hedley, reported details of this example of fraud in the Journal of American Medical Association of July 3, 1937. An important editorial on the subject appeared in the same issue.)

*Remarks in Conclusion*I. *Mortality Incidence*

## A. Organic Heart Disease

## 1. In general

- a. Over 45 per cent all deaths among Metropolitan

## SIXTIETH ANNUAL MEETING

- Life policyholders due to organic disease of heart
- b. Over 600,000 such deaths in the U. S. annually
- c. Becomes greatest cause of death at age 45
- d. Increasing importance in older age groups, population gradually aging

2. Coronary Disease
  - a. Some believe slight relative increase in incidence of coronary disease
  - b. One authority (8) estimates 400,000 deaths in U. S. annually from coronary type
  - c. More important in age group above 40 years
3. Rheumatic Heart Disease
  - a. Greater cause under age 40 years

*II. Life Insurance Medical Research Fund*

- A. Operating over six years
- B. Total nearly \$4,000,000 since 1945
- C. \$600,000 available annually from contributing companies
- D. Grants and fellowships available in fields of physiology, pathology, dealing with diseases of heart and arteries, which cause nearly half all deaths in U. S.
- E. Insurance companies excellent source of statistical material on mortality

*III. Examiner Income from Insurance Companies*

- A. Survey by insurance companies (9) results brought to attention of medical schools, average year (1936) total income of doctors \$850,000,000
- B. One quarter that amount, or \$200,000,000, paid to doctors in fees by insurance companies, (limited to doctors available for insurance work)
- C. Included compensation insurance, fraternal organizations, state insurers, self carriers, contract practice, group and industrial payments, and medical examinations on life insurance applicants
- D. Since then volume life insurance in force greater by more than 100 per cent (now \$243 billions)

- E. Probable that income of doctors from insurance industry also increased
- F. Importance of these figures to medical profession easily apparent

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PRESIDENT YLVISAKER — Thank you, Dr. Gudger. The outline of your discussion of our cardiovascular problems with our medical examiners will be most helpful to us.

Mr. James Andrews, Jr., Director of Health Insurance of the Life Insurance Association of America, will discuss some of the problems with which we are all confronted in group hospitalization and group and individual sickness and accident insurance. You have all received a letter from Mr. Andrews sent you at my request in order that you would have some understanding of the problems which he will discuss with us. Mr. Andrews.

## RELATIONSHIPS BETWEEN THE MEDICAL PROFESSION AND THE HEALTH INSURANCE COUNCIL

JAMES ANDREWS, JR.

*Director of Health Insurance  
Life Insurance Association of America*

It might be well to begin with an incident associated with my admission to the bar in Philadelphia when I was dismayed to find the low esteem in which some lawyers were held in that community. This was brought home to me in newspaper cartoons about lawyers. Every time one of these unfavorable cartoons appeared in the Philadelphia papers the Bar Association would send a letter of objection to the local newspaper which would place it discreetly in the letter column to be read by the more literate members of the community.

The point to all of this is that perhaps 5 per cent of the lawyers in Philadelphia needed to be castigated, but most of them were fine professional people.

We have somewhat the same problem in the accident and health insurance business. Sometimes when I address a medical or hospital association, I give the following little example to illustrate the type of company I do *not* represent.

A certain insurance agent in the accident-health field was trying very hard to sell a policy to his prospect. He said, "This is a wonderful policy. It pays you \$100,000 if you are killed by a buffalo."

The prospect said, "Well, that's not likely to happen; there are not many buffaloes left in the United States."

"Well," the agent said, "it pays you \$200,000 if you are killed by an automobile."

"That's more like it. Tell me more about this policy. Does it have any restrictions or exclusions in it?"

## MEDICAL PROFESSION, HEALTH INSURANCE 163

"Well," the agent said, "it just has one restriction—the automobile has to be driven by a buffalo."

Unfortunately, if people get an adverse opinion of accident-health insurance, I find that they have a rather poor opinion of insurance generally. That is, the things which affect the individual-accident-health field also affect the group-accident-health field, and in general they also affect the life insurance business in its public relations.

There is an attitude among the public—and the doctors in this respect are just normal segments of the public—that we do not pay claims. On the contrary, in the group business which I represent directly, group accident-health claims are paid very promptly. We have demonstrated this in a number of programs set up with hospital associations over the country which have been eminently satisfactory to them. Part of this attitude, I think, arises from the fact that people have an idea that insurance is a very simple business, that it is just a process of taking in some money and paying out some money. I am sure this is true also of the doctors in the general population. They do not realize that there is a complex financial system behind every insurance organization. While the life insurance field is much more complex in that respect than the accident-health field, we do have the same susceptibility to misrepresentation by people who do not understand our business. There seems to be a general feeling that insurance companies have a great deal of money and are therefore indistinguishable from purely philanthropic institutions. The exposure of the industry to this belief is well illustrated in a recent experiment by one of the companies represented here today.

This company was conscious of the fact that one of the great gaps in accident-health insurance was in the catastrophic medical field. There was a great deal of hospitalization insurance written for short-run periods. There was much surgical insurance written for surgical operations. There was a certain amount of home and office calls for medical services written, but if a patient incurred many thousands

of dollars worth of medical expenses, for example, within a given year two or three members of the family were ill, there was not any very effective coverage available. The company is therefore experimenting in the individual field with a policy which will pay up to \$5,000 in catastrophic medical expenses.

For self-protection they use a deductible provision. This excludes payment for the first \$100 or \$300, depending on the deductible provision the policyholder chooses. Then, when expenses exceed the \$300 deductible amount, the patient pays 25 cents out of each dollar. The reason for this is that the patient and the doctor, between them, are in a position to abuse this type of insurance unless there is some control. The patient thus has an interest in keeping low the cost of his illness.

That means that in the accident-health field we have a real problem with the medical profession. But it is not always the doctor's fault. The patient insists on the doctor's cooperation and says, "I have this insurance; I might as well use it. I paid for it."

It is the same attitude that would wreck a government insurance system in this field. We are vulnerable in voluntary insurance for much the same reason, only to a much less degree, of course. In this respect we have a really broad function in bringing home to doctors the elements that are necessary to make the accident-health insurance business function successfully.

I wish to point out, though, certain impediments in our own business which we must remove in order to talk effectively with the doctors and the hospitals. I distributed to this membership an account of the hospital admissions plan technic which was developed in the following manner.

When we first met with the American Hospital Association, we said, "What are your complaints against the accident-health business?"

They said, "First of all, when a man comes into the hospital we do not know what his insurance coverage is. We cannot

## MEDICAL PROFESSION, HEALTH INSURANCE 165

understand the contract. A few hospitals have had to employ people just to review policies, which is very burdensome."

"Second, we do not know whether or not the policy is in force."

"Third, we do not know whether we will be paid directly if we give this patient credit for his insurance."

"Lastly, we are tired of filling out so many different claim blanks."

As a result of these comments, the Health Insurance Council developed a system which is now in effect in nineteen different areas of the United States and which takes care of all these objections. This is operating, at the moment, only in the group field, although we are exploring the same problem in the individual field.

The employer is the key. The employer fills out a certification of benefits which he hands to the employee. This certification tells, in layman's phraseology, exactly what the benefits are. It is a short and simple document.

In group insurance, there are practically no exclusions. Consequently the table of benefits is very simple. The employer signs the blank and indicates where it is to be returned. He gives it to the prospective patient, in duplicate. The patient carries it with him to the hospital, in duplicate. The hospital accepts it. On the other side there is a uniform assignment blank which every company in the business will accept. If the patient wants credit, he signs accordingly. Then, above that is a uniform claim blank supplying enough claim information to satisfy the requirements of every company in the business. This assures the hospital of prompt, direct payment and alleviates the clerical problem of filling out claim forms.

Most important, I think, it makes clear to the person immediately when he needs to use the insurance what his insurance provides. We have had a lot of criticism in that when our salesmen talk to people about insurance they do not

give them the proper information concerning their coverages. Before this system was installed, people were told that their insurance was no good, and they were forced to make a cash down payment before being admitted to the hospital. Hospitals have been quite satisfied with the system, and it has been a stepping stone to better relations with insurance companies.

I shall take a moment to describe an item which is of more direct interest to you.

The Health Insurance Council has set up surgical committees which are more or less on a state basis. These committees confer with medical societies, when requested, on the formulation of voluntary prepayment surgical plans sponsored by medical societies and underwritten by insurance companies. Doctors, you see, have intensified their interest in this field because of Mr. Oscar Ewing's proposals in Washington. Thanks to Mr. Ewing, we have sold greater amounts of insurance because everybody is very health-conscious today.

We are now trying to improve the voluntary system by discussions with medical societies about prepaid surgical plans which they themselves promulgate, but with our advice.

The main purposes of these programs are two. One is that in our files we find many instances of a doctor's charging more just because a person has insurance. In effect, he is really helping to destroy the voluntary system. I do not mean that this is all intentional. Certainly most of the instances are not those which would be taken to a grievance committee. They are not very much out of line, but let me give an illustration that came to our attention from one company.

This is one of those coincidences in which two people are in exactly the same economic circumstances. The two women concerned were sisters. These sisters happened to have the same operation performed by the same surgeon within three or four months of each other. The sister who had Blue Shield coverage was charged \$150 for that operation; the one who had insurance coverage was charged \$300.

## MEDICAL PROFESSION, HEALTH INSURANCE 167

We went to the doctors and described our complaint. They said, "Yes, but look at your policies; you do not expect us to accept these benefits in full payment, do you?" So, we said to them, "Very well, you construct a schedule which you think would be fair for the low income group which you would be willing to accept in full payment for a specified low income group."

They, with their various specialist groups, constructed a surgical schedule. When they were through, they sent it to the members of their society, and the members signed up with their own medical society. They did not sign up with the insurance companies. There is a written agreement between each member and his society that he will accept a certain list of surgical fees in full payment for service to the people below the specific income limit.

Then an invitation was sent out by the medical societies to the insurance companies authorized to do business in the state, and the insurance companies were asked to participate in the program. They do so by placing the doctors' schedule in their policies. When the schedule is sold to an employer for his employees (or even to individuals), the certificate holders in the group have the assurance that their surgical fees will be paid in full if they are below this income limit.

The concept of these plans has been criticized in some quarters by doctors as being much like that proposed by the government, but we believe strongly that unless the doctors do something about making their fees definite, the chances of the government's program coming back to haunt us are great. Congress might not pass the Ewing program in its original form, but one must remember that there are many sorts of compromise bills available. Actually, we have sold a good deal of surgical insurance without the doctor's assistance. There are over 50 million people covered by Blue Shield and by our own companies, and I think the coverage is about equally divided.

One of the by-products of bringing these groups together is an item like the one which appeared in the *Tennessee Medical*

*Journal.* It was written by the president of the Tennessee Medical Association. In this state there are 300,000 people covered by one of the plans that I have just described. He wrote as follows:

"A doctor in Nashville made a significant appeal at the recently called session of the House of Delegates of our association. The doctor said that voluntary health insurance plans would not and could not survive unless doctors, patients, and hospital administrators stopped certain abuses of hospitalization."

His appeal at the meeting was directed to the medical profession. He cited certain abuses which doctors sometimes inflict upon hospitalization plans, such as admitting patients to hospitals for conditions which do not require hospitalization.

Your Association, which is a member of the Health Insurance Council, could be of some assistance to us by having its members pass on word that the situation is ripe in different states for discussions in which we might participate. We are not promoting these plans, but once in a while and as recently as six months ago, we were called to Cleveland after the plan was already constructed. The doctors desperately wanted us there to compete with Blue Shield, but they had already constructed their plan before we arrived. We need to arrive on the scene early, and sometimes we need some support.

In North Carolina a year and a half ago the House of Delegates had passed one of these programs. We had been there a number of times with our industrial committee. The House of Delegates had passed the program and later some of the national Blue Shield advisors, who do not like to be associated with us, came to North Carolina and convinced the doctors to reverse their previous action. We were summarily tossed out. Subsequently, I discovered that one of the life insurance medical directors happened to be president of a county medical society. It would have been of great

## MEDICAL PROFESSION, HEALTH INSURANCE 169

help, had we known of this obviously important fact when we went there.

Thank you very much for your kind attention and interest.

**PRESIDENT YLVISAKER** — I should like to tell you that our Association has two representatives on the Health Insurance Council, Dr. Ungerleider and Dr. Filson. Does either of you want to comment at this time?

**DR. HARRY E. UNGERLEIDER** — I should like to tell you this much. The best thing this Health Insurance Council has done for the life insurance companies is to improve relations with the medical profession. This Health Insurance Council and its predecessor body came into being some five years ago. Since that time there have been various meetings with the American Medical Association and the state medical associations and with smaller groups. We have been able to tell these small groups what the life insurance companies, and particularly the group life insurance companies concerned with health and accident work, are doing.

It sometimes has been a little rough, but our efforts have been rewarded. I believe that the Health Insurance Council is probably the best public relations agency the insurance companies have with the medical profession. I therefore ask your support of this Health Insurance Council. If you will write to Dr. Filson, to Mr. Andrews, or to me, we will do everything we can to help in any problem you may have.

**DR. RALPH FILSON** — Mr. President, Members of the Association: I shall be very brief in the few comments that I would like to offer at this time. It has been my privilege to represent this Association on the Health Insurance Council only for the last couple of years, and I have greatly enjoyed and benefited from the opportunity of observing the deliberations and activities of the Council.

I believe it is particularly timely for us all to have had the opportunity of hearing what Mr. Andrews has had to say to us this morning. My thought is this. In this membership there are increasing numbers who have active business in-

terests in the accident and health field. We all, I believe, have interests beyond those businesses with which we are associated. I believe the membership of this Association should recognize that now, by virtue of a closer liaison between the insurance industry and our own profession, we have an opportunity to promote, through our companies and within our local medical groups, the wider use of insurance to meet the challenge of compulsory health insurance.

PRESIDENT YLVISAKER—I want to thank Mr. Andrews for coming here to our meeting, and Dr. Ungerleider and Dr. Filson for their work on the committee. I am sure we can all feel that we are well represented and that these gentlemen will help with any problems that arise in connection with hospitalization and health and accident insurance.

In my remarks yesterday I referred to the fact that we in our work see impairments in individuals before they themselves are aware of them. I also emphasized the fact that when we learn of those impairments it gives us the responsibility of doing what we can to eliminate them. This problem brings us in contact with the public health field of medicine, and our program committee felt that we should devote a part of our program at this meeting to our public health relationships and to our responsibilities in that connection. Public health has contributed much to our work, and perhaps we can contribute something to public health medicine.

In arranging for this part of our program, we have tried to bring to you some of our leading public health authorities. Our first speaker was graduated from the University of Minnesota and entered the public health field in Minnesota. He advanced into the United States Public Health Service where he became chief of the Division of Tuberculosis. When New York State needed a new Commissioner of Health he was selected. He has become a world figure in public health, and it is a privilege to have him with us. It gives me pleasure to introduce to you Dr. Herman Hilleboe, Commissioner of Health for the State of New York.

## THE PUBLIC HEALTH SITUATION TODAY PUBLIC HEALTH AND CIVIL DEFENSE

HERMAN E. HILLEBOE, M. D.

*Commissioner of Health  
New York State*

This morning the subject that I have to present is a very serious one. I am going to talk predominantly about civil defense and the relationships between civil defense and public health. As many of you realize, civil defense is nothing more than an exercise in preventive medicine and public health.

What we are going to do and are now trying to do can be stated very simply. It comes down to this: for the purpose of maintaining manpower and morale, the mission of civil defense medical activities is to prevent death, to reduce disability and to relieve suffering following the explosion of an atomic bomb.

The military authorities have given us rather specific information that the probabilities of an atomic bomb's being launched in this country are very definite. I am not going to question the basis of their prognostication, but I will say that these people are usually right. I happen to have had the privilege of being in Korea in July of this year. I had an opportunity to go up to the front with some of General Van Fleet's staff and to see some of the operations that are taking place. Certainly when one gets that close to the realities of war, one can appreciate that predictions based on any probability, whether it is one in one million or one in one thousand, must be given serious attention.

The important thing for us in New York is the definite possibility of an atomic bomb's being dropped. Accordingly, we must set up our plans on the basis of this probability and attempt to prevent as many disabilities and deaths as we can. Bear in mind that the population of New York State is something over 15 million, that we have many target cities

or potential target areas in this state, and that our planning must encompass cities of various sizes. Certainly the planning that we carry out for New York City is going to be quite different from that which we carry out for Syracuse or Buffalo or the Albany-Schenectady area. Therefore, in our planning, from a medical viewpoint, we must bear in mind many of the factors that we learn from a study of the epidemiology of various diseases in public health. We must bear in mind the concentration of population and also the concentration of industries.

Whenever we start a new project in public health, or in medicine for that matter, we at once look back in the literature to see what has been accomplished and what is known in this particular field. In civil defense during the war we had the experience of strategic bombing effects in England and Germany. We have all the reports, of course, of strategic bombing in Japan. We have investigations of psychiatrists who studied the population groups after the bombing and attempted to find out the effect of bombing on the mental attitudes of the people.

In spite of the fact that we have a vast amount of firsthand information on the effect of high explosive bombing used in the last war, we have no such information on an atomic bomb attack.

On July 21 of this year I visited Hiroshima in Japan. I had an opportunity to talk with some of the physicians who were there when the bomb dropped. It is rather amazing to see that after six short years, all of the buildings with the exception of two have been rebuilt. Those of you who have seen pictures of Hiroshima sometime shortly after the bomb was dropped appreciate that nothing was left within an area of about one square mile. All that area has been rehabilitated. There is no evidence remaining except the one building right near the river where the bomb was exploded about 2,000 feet in the air.

The only building left in its bombed state is an industrial arts museum that was made of concrete reinforced with steel,

and only the shell remains. It remains because the Japanese wished to leave a monument to the atomic bomb that struck their city. Ironically enough, they have called it the "Peace Building." As far as learning, however, what we should do in New York City or New York State if a bomb should drop, there really is not much that we can learn from the Hiroshima or Nagasaki experience. The reason for this is that quite evidently neither of these cities was prepared for the atomic bomb when it was dropped.

You might be interested, also, in knowing that in spite of the fact that an atomic bomb was dropped on Hiroshima, there is no real civil defense program at the present time in that city.

Now we cannot let ourselves labor under the delusion that we might not be hit in this city or that city. In New York State we have taken the attitude that we have to prepare every one of our target areas, and we have to prepare a long time ahead if we are going to be able to reduce the number of deaths and the amount of disability that could occur.

In speaking to some of the physicians in Hiroshima, I got the impression that these men felt that if an opportunity had been provided to prepare for at least two years for the atomic bomb, the number of deaths probably could have been cut down by as much as 75 per cent. The disability could have been reduced, also, because what happened is what usually happens in an unexpected catastrophe: many people who are severely injured cannot be given attention immediately. The result is that infections set in, complications occur, and I do not need to tell you what happens in terms of disabling illness.

In our own planning in New York State, we received a good bit of information from England because of the many bomb hits that occurred over there during the last war. Three things are pressed home when we look at the British experience. In the summer of 1950, in connection with some World Health Organization activities, I spent two weeks in England and went to some of their training schools and

visited a few centers where bombing had occurred. The English have taught us that three things are exceedingly important. First of all, it is quite necessary to carry out detailed planning for civil defense a long time before the event. In our country we are hoping that we might have as long as two years from the time we started our formal activities in May of 1951. In England, the people felt that if they had had more time to prepare for the bombing attacks they could have reduced the number of deaths and the amount of disability.

The second point is a very important one and involves the subject of training. The English realize that to carry out an effective program in the event bombing occurs one must carry on extensive and prolonged training. By training, we mean much more than simply listening to a few lectures on civil defense, reading a few pamphlets and seeing perhaps a few films. Training to the English is very realistic.

I went up to Easingwold, which is one of the suburbs not far from Bristol, where the rescue school for English workers is established. At Easingwold about thirty strong, husky men from the provinces were brought in every three weeks, and these men were given an intensive course of training in rescue work, including about sixty hours of first aid. At the end of the first week the realistic training begins. At Easingwold there are several structures that have been constructed for the purpose of being blown up. These buildings are so fixed that underneath there are tunnels, places where decoys can be inserted so that when the teams go out they can actually rescue live decoys.

One night about eleven o'clock we heard a terrific explosion. We looked out the window and saw a flash of fire. The second story of one of these buildings started going skyward. The eight men on the rescue team on duty that night jumped into their clothes, went over to the truck and drove to the bombed building.

In the rain they started digging under the debris to get out some of the people who were supposedly injured. The

English in their realism hired some of the retired army sergeants and by giving them a little extra money induced them to hide under a building when it was actually blown up and to be there when the rescue workers searched the ruins. Various injuries were simulated with makeup. In order to show a fracture they took the bone out of the leg of a cow and made it stick out of the pants leg of the supposedly-injured individual. When the rescuer got into the ruins he had to give first aid, carry the person out, and then render additional first aid when he got the person out of the building.

Now, such realistic training is time-consuming and it is expensive. But it is the only possible way to teach people how to do things in the event of serious bombing.

The third point that we learned from the English is that of morale. We ordinarily do not talk much about morale in our country because we take it for granted in most communities. But, the English have learned that during a bombing, unless provision has been made for doing something for morale, panic develops. Panic could kill more people than the bomb itself. It is a very important lesson to learn. Hence, the English have attempted to develop group leaders in every block of every city. There might be a family leader as well as a block leader, and that individual's job is to teach each adult in the block something about first aid and something about bombing, so that if a bomb does come the people will know what to expect. Of course, calmness among large numbers of persons results only from calmness among small groups.

In the Army there is little concern about panic because the men are fully trained. They are disciplined and are used to taking orders. But among the civilian population we have quite a different situation. Therefore, in planning for civil defense we must be exceedingly careful to pay attention to the effect of a bomb on the mental attitudes of people and the way in which they will react under mental strain and physical stress.

In our own state of New York, the Health Department has been given the responsibility of statewide planning for the medical and public health activities in civil defense. We have taken the attitude that the atomic bomb is a realistic fact, and that the Office of Medical Defense will become a permanent part of our Health Department. I think you gentlemen should assume the same attitude, because in the state of living that we have at the present time, it looks as if we will have to be prepared if we wish to survive.

Therefore, in our own Health Department, we have requested state funds in considerable amounts in order to set up a permanent program. Late in 1950 we had an appropriation of a half million dollars to get under way with our program of medical defense against the atomic bomb. The session of the Legislature which ended on March 31, 1951, appropriated over 12 million dollars just for the medical aspects of civil defense.

I mention these figures to give you some idea of the seriousness of the situation. Our total budget in the Health Department for the present year is 40 million dollars. To that has been added the 12 million dollars of state funds and 5 million dollars of federal funds for the sole purpose of building up our medical defense against the atomic bomb. It comprises now almost one quarter of our total Health Department budget. Although we have some full-time men and women assigned to this program, the fact remains that a considerable amount of the time of all personnel in the department is spent on the problems of medical defense.

There are one or two things that might be worth mentioning in terms of the development of this program. In the first place, we recognize the importance of bringing the practicing medical profession into the program of medical defense. It was necessary, in addition to the administrative people in this field, to have arrangements made to get the advice of the practicing physicians. In the final analysis, the work that they will do with the injured is going to determine whether or not we will reduce deaths and disability.

We had the problem of setting up advisory committees to give us advice and counsel on this particular problem. At present there is a tendency, I think in public health and in other fields of medicine as well, to appoint a committee whenever one gets into any trouble. The greater the trouble, the greater the number of committees appointed. Soon there are so many committees there is not time for the executives to do any work. We must, therefore, be cautious about setting up too many advisory groups to cope with a problem as large as this. We are very fortunate in New York State in having a Committee on Emergency Medical Preparedness of the State Medical Society. We called this group together at one of our monthly meetings and asked their advice on the best way to use their membership without taking too much of their time.

We agreed that two committees would be sufficient to give us the advice we needed in developing our program. I think this technique has been very useful now that we have had it for about a year, and I should like to describe it briefly.

First, we set up a medical advisory committee on civil defense. That medical advisory committee consisted of the officers of the State Medical Society and the chairmen of the principal committees concerned with civil defense and public health. Those included the chairmen of the Emergency Medical Preparedness Committee, the Blood Bank Committee and the Committee on Public Health and Education. I mention a few of these to give you some idea of the type of skills brought into the medical advisory committee. It is apparent that if we needed some special help in the field of blood banks we would not set up a new committee but would ask the chairman of our committee on blood to bring together a small group of experts to get their advice on the problems presented. Thus we have a closely knit group which does not require the time or presence of large numbers of individuals. These men meet with us for half a day the second Wednesday of every month and we go over our problems at that time.

I should like to give you an example of one of the other things this committee has done. We needed to set up routine procedures on the treatment of burns, fractures, bruises and injuries, shock, and on the use of blood. Each of these problems as you know is difficult in itself. We recognized that we might have to carry on treatment of this kind in different parts of the state, such as Buffalo, Rochester, Syracuse, Albany, and New York City, as well as in other metropolitan areas.

When we came to treating burns we encountered the problem of storing millions of dollars worth of bandages, and I mean millions of dollars worth of bandages, because if we have fifty or one hundred thousand burn cases they would require a lot of medical supplies. We had the question of purchasing equipment in addition to medical supplies and we had also the question of uniform training in the use of these supplies and equipment; we obviously must have uniform training methods for the physicians of the state who are going to participate.

It seemed logical to bring together representatives from the nine medical schools in New York State to discuss these problems. The chairman of the Emergency Medical Preparedness Committee of the State Medical Society called together the deans of the nine medical schools. I think it is one of the few times in the history of New York State that the nine schools have been represented in one room at one time. Not only did they get together and agree to follow through, but they suggested that a specialist in the treatment of burns from each of the schools be brought to one place, and a specialist in the treatment of fractures, and so on down the line in the different groups that I mentioned. This was for the purpose of agreeing on uniform methods of treatment so that we could begin to stockpile supplies and in every way conserve manpower and money.

We arranged to have the facilities of the New York Academy of Medicine available for the forty-five different specialists that day. We broke up into five groups, with nine men

in each of the groups. There were nine specialists on burns, nine on shock, nine on fractures, and so forth. I sat in on the burns panel at that first meeting and I can assure you that it was quite interesting, because those who treat burns have some very individualistic ideas about how burns should be treated.

Our problem was to eliminate as many varieties of treatment as possible, and preferably all but one. By the end of the day there was recognition that there must eventually be some agreement on common ground. This perhaps sounds like over-simplification, and it is in a sense, because the treatment of burns is exceedingly complicated in many cases. But, the fact remains that in the first twenty-four hours after a bomb burst there are certain things that can be done, and in the first four to seven days there are more complex things than can be done.

What are the minimum procedures necessary to save the lives of burn victims? It was rather amazing to us that, as a result of this first session and the second session held two weeks later, the group arrived at one treatment for burns. This treatment was accepted by nine specialists from the nine medical schools. Likewise the special group agreed upon a very short and concise summary of the emergency treatment of fractures.

During the third session we called in the chairmen of the groups and asked them to check for overlaps in the treatment of shock, fractures and burns so that in the final document there would be no duplication and there would be consistency in the whole program.

This is the way in which this medical advisory committee has operated, and as a result we were able to get out a concise statement for the use of all of our physicians in the state, including New York City. This was published in our official publication, *Health News*, in June 1951 and was sent to every physician practicing in the state of New York and also to all health officers. Incidentally, you might be interested in this particular document because it is an example, in

the field of civil defense, of unanimity of opinion on a subject that involves both public health and clinical medicine.

The other advisory group that we developed was the advisory body called the Health Resources Council. We recognized that there would be people in the hospital field, the nursing field, and the pharmaceutical field, and about twenty-seven others, with contributions to make. In addition, therefore, to our Medical Advisory Committee, we established a Health Resources Council with the principal officers of the state organizations as the representatives. For example, the secretary of the New York Hospital Association was asked to serve, as was the secretary of the New York Pharmaceutical Association. All of the professions allied to medicine and public health were invited to send representatives to serve on the Council in this way.

This Health Resources Council was called in mainly to give us help. In other words, we asked, "How can you participate in the civil defense program, particularly in the medical field?" In turn, we attempted to interpret to them the size of the problem, what we were doing about it, and what we thought the various organizations could contribute. This group helps us to get across the message of civil defense and the importance of doing something about it to all of the professional groups in the state.

These are the only two advisory groups that we are using in our civil defense program. I have had requests for a number of committees to be formed. The step that was taken last year has proved to be a sound one, and it is worth mentioning because it is a principle in public health that we must conserve our limited energies in devising and executing a definite plan for the medical activities in civil defense.

We need to discuss perhaps a few of the specific problems that we have encountered in developing our program of medical defense against atomic bombs. One of these is in the field of personnel and training. First of all, in order to orient the physicians of the state, we agreed, with the help of our advisors, to set up a one-day orientation course for

physicians. This consisted of two lectures of three hours each. In that endeavor we tried to bring in all the medical societies throughout the state and all of the practicing physicians so we could tell them first, what are the medical effects of the atomic bomb; second, what is the plan of the state of New York; third, what should be done in their local communities; and fourth, how they should participate in the program.

It is difficult to get physicians to come out to hear about medical defense or public health or something that is not directly related to the patients who come into their offices. They are so absorbed in their practices, and understandably so, that if we want to reach them concerning something outside their particular fields, we find that they are not especially interested. We were confronted with the problem of interesting specialists and general practitioners in this program which is voluntary on their part and on our part, of course, as well. We tried out different technics of reaching large groups of physicians. With one medical society, we felt we would see what we could do by putting out the usual bulletin saying that there was to be a meeting on the medical aspects of civil defense. The membership included some four hundred physicians. At the first meeting, I recall seventeen physicians showed up. Obviously that was not worth much to civil defense.

The next time we came back to that same place we went to the medical societies' auxiliaries, the newspapers, and every group in the community that we could contact. We had the wives of physicians call up the wives of other physicians and check back and forth to get definite replies on their husbands' attendance. As a result we reached, at the second meeting, 80 per cent of the membership of that particular county medical society. The secretary wrote and told me later that in all the years of the existence of the society this was the greatest turn-out the society had ever had.

Of course, the future training of physicians in civil defense will be more detailed than in the courses just mentioned. We have to give them actual exercises in setting up first aid

stations and emergency hospitals. They must become familiar with the part they will play in the first aid stations, in the emergency stations, improvised hospitals, and in permanent hospitals. We are proceeding with definite programs along those lines.

One of the other things that we did of great importance was the developing of the training program for adults in first aid, or self-help and neighbor-help, as we call it. I venture to say you might think it a simple thing to get individuals to come out and take first aid courses, but those of you who have had anything to do with the Red Cross program appreciate the difficulties of asking an adult member of a family to spend twenty-two hours of his evenings and weekends learning about first aid. We went to the Red Cross and asked them if they would set up for us a short course instead of their regular twenty-two-hour course. It was not possible for the Red Cross to set up a short course. The Red Cross thought a short course might interfere with their regular training courses, but we knew that if we were going to reach a large number of people we had to curtail the amount of time invested. We developed a series of demonstrations and lectures that involved four sessions of two hours each. In order to avoid conflict with the Red Cross, we did not call it a first aid course. From the English we took the term "self-help" and added our own of "neighbor-help". And so we set up a course in self-help and neighbor-help.

By developing a simple little manual and by having teaching methods that include such items as flip charts, splints, and bandages, we were able to get under way within a period of three months and started training instructors throughout the entire state of New York in the important problem of first aid. Ordinarily in a public health enterprise of this type we would like to have a year to prepare the booklet, in order to do the job properly. We had to do it in six weeks. It was necessary to break all the rules of government purchasing in order to let the contracts on these booklets the first time we ordered them. We ordered one million copies and are extending their use every day. In civil defense one must

cut corners and at the same time attempt in every way possible to maintain the quality that is absolutely essential in any medical activity.

I shall not go into the details of our training program for nurses, for messengers, for litter bearers, or for rescue workers, but each of these special groups must have a training program and an exercise program. We consider training one of the most important parts of the entire medical defense activity.

When we come to the question of medical supplies and equipment, we meet some very serious obstructions. One of the things needed, obviously, is morphine. We shall be purchasing morphine in 100,000 dozen lots. What are we going to buy? Quarter grains? Sixth grains? Or half grains? Or are we going to buy several different doses? Are we going to put it in ampules or are we going to put it in syrettes. It has been necessary, of course, to look ahead a long time because the storage factor is an important one. Again, we brought in the experts from our medical schools and got the best advice available. On the basis of that advice we went ahead and set up plans to purchase it. Again, it is not so simple as putting in an order to a drug firm and storing the morphine in a certain place. First of all, we must have permission from the Federal Bureau of Narcotics to buy large quantities of morphine. We had to give assurance that it would be stored in such a place that it would not get into illicit channels, because the quantity that we were buying at forty cents a dose could easily bring five dollars on the illicit drug market; there are groups that would not hesitate to kill quite a few state employees in order to get at that amount of morphine. We had to assure the Federal Bureau of Narcotics that it would be stored in one of our state prisons where there are several sets of locked doors. The morphine is kept in a vault; we have bonded people taking care of it, and we have an alarm system set up so that the state police would be alerted in case anyone was bold enough to try to steal our supplies.

Another problem of great magnitude has been the matter of bandages for the treatment of burns. There has been some discussion by the National Research Council and also by the Armed Forces as to the best way to treat burns immediately after they occur, as well as the problem of the treatment in the twenty-four to seventy-two to ninety-six hours thereafter. Our burn panel suggested that we do away with all medication as far as local applications are concerned, and simply use the Army type of large padded bandage. This meant that instead of ordering different types of ointments and such things as liquid petrolatum we needed to order only burn dressings. By the use of bandages and morphine and blood, of course, for supportive treatment, we could limit orders to those things that were absolutely necessary. The question of the size of the bandage was an important one.

We investigated the question of treatment of burns after the immediate period. I had the opportunity to visit some of the hospitals in Tokyo where the burn cases from the Korean battle front are treated. Those of you who have been there know that in the Tokyo General Hospital there is a thousand-bed unit, and many of those beds are used in the treatment of burn cases. I think we can make good use of the experience in Korea and Japan in the treatment of burns after the immediate period. The army group has had good results with the old method of the open treatment of burns. If this works—and it appears to work—it is going to save us hundreds of thousands of dollars and certainly will be helpful in the immediate days following the explosion of an atomic bomb.

I went into a burn ward in the Tokyo General Hospital and saw about thirty cases that had been treated within the last two to twenty-one days. I had an opportunity one morning to see six fresh cases come in and to see them handled in the operating room. A quarter grain of morphine intravenously is given preoperatively, and during a period of about fifteen to twenty minutes the surgeon, under sterile technic, simply scrubs the burned area with sterile gauze and one of the detergents. All the loose blisters are scrubbed

off. The burn is left denuded and the patient is wheeled back to the ward. He is then put on clean warm sheets and left exposed, and in about twenty-four to forty-eight hours a crust forms. This crust has pretty much the effect of the old tannic acid we previously used, with the exception that the tannic acid was toxic in many cases and had to be discontinued.

This natural crust apparently serves several purposes; it keeps out infection, avoids contact with the air, and obviates pain. Over a period of about twenty-one days, the partial-thickness burns heal very nicely; the full-thickness burns can have skin grafts after the other burns are well on the way to healing.

You appreciate, of course, that this treatment must be limited to first and second degree burns. It will not take care of the full-thickness burns.

This treatment of burns is highly important to us. We have now requested our panel on burns to review the Army procedure and to consider its use in medical defense. Again, this is an example of one of at least twenty different things in which we must first of all have concurrence from the practicing physicians as to the treatment of choice. Then the question of training physicians to follow this treatment is an important consideration. Finally, there is the question of stockpiling the necessary supplies and equipment.

The problem of blood is a very serious one. We face the question of whether to use whole blood or blood derivatives and blood substitutes. It is one problem on which we have used our medical advisory committee to very good advantage. We plan to give predominant attention to whole blood because in the severe injuries of the type with which we will have to deal, we shall need blood sooner or later. The more cases in which we can use it sooner, the fewer cases we will have to use it in later.

Again, in developing a blood program, we have had to call on our 250-odd laboratories throughout the state to train additional people in blood grouping and processing, and to

increase the number of individuals on the donor lists. In the first hours of any atomic attack, we would have to limit our blood to Group O type.

We shall be concerned with keeping people alive, and Group O blood can be used generally with minimum reactions.

Biological warfare and chemical warfare also absorb our attention. There is not too much known about biological warfare. We do have some information on the types of epidemics that might hit us, either intentionally or unintentionally and we are attempting through our epidemiological service to be prepared for any unusual incident that might occur, and to isolate that area for investigation. I will not go into these problems at this time save to mention that we have called upon our medical advisory committee groups to bring their best minds together and to give us the kind of advice that is necessary to set up a sound program.

You may wonder why I have not mentioned radiation burns in the discussion. I have not mentioned it for the specific reason that experience in Hiroshima and in Nagasaki has shown us that perhaps less than 15 per cent of the injuries will be radiation injuries.

There is no point in giving a lot of blood and a lot of medical care—taking care of the fractures and the burns—of people in the immediate area who have had more than 600 roentgens of radiation. They are going to die in spite of anything that we do, and so we simply cannot do more than try to allay their suffering. We have to make them comfortable and alleviate their pain in every way possible, but we cannot concentrate our attention on the people who have had excessive doses of radiation. Our efforts must be made available to the 85 per cent of the wounded for whom there is some hope of recovery.

That means that most of our energy has been directed toward setting up programs for persons least exposed to radiation hazards. If we were to give 50 per cent of our attention to the 15 per cent who are badly burned, we would lose a

good many of the wounded among the 85 per cent who do not have severe radiation burns. We are trying to give priority to the people who can be saved by emergency care.

Considerable quantities of atropine are being stored along with our morphine. We are able to store atropine at strategic places because, first of all there is no addiction problem there, and second, if atropine is going to be of any benefit it will be in the first few minutes and not in the first few days; so we are attempting to take advantage of that fact.

The last thing I want to mention is the consideration of mortuary services. After people are dead, of course, the physician cannot be concerned about them. However, because of the fact that the physician will have to determine whether or not an individual is dead, because of the fact that he is accustomed to dealing with the injured and the dead, it is natural that the civil defense authority would place upon our shoulders the responsibility of the collection, identification, and disposal of the dead. This is a very serious problem because it can interfere with medical care unless there is an efficient means of removing, identifying, and disposing of the dead. The complications that will come in any community are tremendous.

The problem of civil defense is a very serious one. We are attempting to meet it in a systematic way. It is truly an exercise in preventive medicine. I think that you gentlemen with your knowledge of preventive medicine can certainly be of help in your own communities. Your knowledge and experience in the field in which you are working should be very useful to your local civil defense group. I hope as you go back to your own communities that you will find out about medical advisory committees, find some way of getting into that group and giving these men in the local communities the benefit of your experience and knowledge. If an atomic bomb should be dropped over New York City today, next month, next year, imagine the immensity of the catastrophe, if you can, of several hundred thousand injured people, perhaps the same number of dead. It will require

all the resources of the state of New York, the neighboring states, and everyone within reach.

We feel that civil defense is something that has become a permanent part of our way of living. In the Health Department we have taken the attitude that we need to do double duty. We are giving all the time we can, including evenings and Saturdays. We do not like it. Our wives and children do not like it, but I think we have no choice. And so, in conclusion, may I ask that as you go back to your own communities, you find out what is being done about the medical aspects of civil defense, and offer your services. If everyone will help, then I think we have the greatest chance of survival.

**PRESIDENT YLVISAKER**—Dr. Hilleboe, please accept our thanks for coming to our meeting and for impressing us as you have in your talk with our public health and civil defense responsibilities.

Dr. Karl W. Anderson of the Northwestern National Life Insurance Company has just called my attention to the fact that the University of Minnesota College of Medical Sciences at the annual faculty dinner this week, honored outstanding alumni and gave Dr. Hilleboe this citation: "A distinguished graduate of the University of Minnesota, Commissioner of Health of the state of New York, outstanding public health administrator, significant contributor to tuberculosis control." It has been a pleasure for us, Dr. Hilleboe, to have you with us.

We have several members of our group who have devoted themselves to public health activities and who have won distinctions in that field. One of them will discuss the contribution which public health has made to life insurance. The other will tell us what we in our work can do to contribute to our very important public health program.

Dr. George M. Wheatley of the Health and Welfare Department of the Metropolitan Life Insurance Company will discuss "Some Contributions of Public Health to Life Insurance."

## SOME CONTRIBUTIONS OF PUBLIC HEALTH TO LIFE INSURANCE

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It is a great privilege as well as a pleasure to have this opportunity to relate some of the achievements of public health contributing to life insurance. Your training and experience, as well as your position in our business, give you, I like to think, a perspective on human life, individually and in the aggregate, closer to the point of view of the public health official than to the clinician.

By "public health" we mean those organized efforts by health agencies, private and governmental, to wipe out—or to reduce—death and disability from disease and accident and to improve the health of the population by education, research and preventive technics. Two features distinguish public health practice from remedial medicine. In dealing with health problems, public health is concerned primarily with *prevention* rather than treatment; with the *group* rather than the individual, although the individual is coming into his share of attention through periodic examinations. This approach has made it possible to apply the principle of "risk mitigation," a standard practice in other forms of insurance, to life insurance.

### *Past and Present Contributions*

The concept of prevention applied to disease and trauma is the essence of the public health movement in this country. Not only has life conservation been intimately related to the remarkable economic, political and social growth of our people, but today in the fight to keep freedom strong and alive all over the world, our public health "know how" has become one of the most valued products that we export.

Probably no business has benefited more directly from the application of this idea than has life insurance. On the other hand, few businesses have so actively aided the expansion of public health activities. It should be emphasized, too, that this mutual advantage has long been recognized by spokesmen for life insurance as well as spokesmen for public health. Because the great saving in mortality has come largely through the conquest of the infectious diseases and the control of epidemics, it may appear to some that the point of diminishing returns has been reached in regard to this beneficial and mutual relationship. It is timely then to review the influence of public health on life insurance and to consider the prospects ahead.

#### *Communicable Diseases*

As just indicated, the obvious and most important impact of public health on our business has been the tremendous improvement in mortality over the past fifty years through the control of the communicable diseases. The facts are so well known as to warrant hardly more than an illustration or two.

The conquest of tuberculosis is perhaps the most striking demonstration of the power of organized effort to combat disease. In 1900, it was the leading cause of death in the general population of the United States with a rate of 195 per 100,000. Among Metropolitan Industrial policyholders, in 1911, the rate was 225 per 100,000. For the country as a whole, the rate now is under 30 deaths per 100,000 and our Industrial policyholder rate this year will probably be below 20. Better personal hygiene, isolation of open cases and specific treatments have been major factors in this conquest. Case finding has been expanded enormously by new technic in mass x-ray examination. In this battle, practicing physicians have been in the front line, but the weapons, the strategy and the tactics have been devised and executed by public health agencies, both official and voluntary.

Diarrhea and enteritis, the scourge of infants, was the third cause of death in the United States in 1900, with a rate of

116 per 100,000. Now it has virtually disappeared as a major cause. In our own Industrial experience, since 1911, the mortality rate has declined 93 per cent. The communicable diseases of childhood as causes of death have almost reached the vanishing point. For example, in New York State, so far this year, not a single diphtheria death has been reported. In this state alone, fifty years ago, there were nearly 3,300 deaths from diphtheria. For the United States as a whole, it is estimated that had the diphtheria death rate of 1900 prevailed in 1950, there would have been 50,000 deaths in that year. The actual number of deaths last year for the entire country was 432. For life insurance, the saving in lives of children has meant, among other advantages, liberalization of benefits.

#### *Maternal Mortality*

Voluntary agencies have taken the initiative in many of these health campaigns. The control over maternal mortality is a good example. Until about 1920, the maternal death rate in this country showed little improvement. At that time, a report by the Public Health Committee of the New York Academy of Medicine showed that two thirds of the maternal deaths in New York City were due to errors of technic or judgment on the part of the attending physician. Medical society committees were promptly formed in the five boroughs to study every maternal death. Within ten years the puerperal death rate in New York City had dropped more than 60 per cent. The Maternity Center Association in cooperation with obstetricians and public health organizations, using this and other studies, has had a strong influence in improving obstetrical practice throughout the country. In 1911, mortality from puerperal diseases among our Industrial policyholders was 20 per 100,000. Last year, it was 1.8, a decline of 91 per cent.

#### *Epidemic Control*

Epidemics such as that experienced with influenza in 1918-19 no longer hold the same threat because of the present level of development of public health organization. The U.S.

Public Health Service, the Rockefeller Institute, and medical and public health centers cooperate on a national and international basis to collect, study, and report local health conditions so that preventive measures can be quickly mobilized if some infectious disease threatens to assume epidemic proportions. For influenza, this includes provision for identifying and typing the virus and preparing, if possible, the appropriate vaccine with the cooperation of the commercial biologic laboratories. The influenza epidemic in England last year was handled in this organized manner. Infantile paralysis is kept under similar study during its seasonal prevalence. The powerful protective forces which public health can quickly mobilize are illustrated by the smallpox outbreak which occurred several years ago in New York City. In spite of some delay in spotting the presence of the disease, a serious epidemic was averted by the promptness with which the population was vaccinated. Under the leadership of the Health Department, all public health and medical resources were quickly brought into action to aid in the vaccination program.

#### *Accident Reduction*

As previously shown, governmental agencies are not the only groups capable of planned and directed effort to improve health. The National Safety Council is a voluntary agency supported by industry, including life insurance companies, which has done for many years an outstanding job of reducing death and disability from accidents. Industry has been largely responsible for the fact that the worker now is far less likely to be injured on the job than he is on the highway or at home. Last year, among Metropolitan Industrial policy-holders, the death rate for home accidents was more than double that for occupational accidents. Through the leadership of the National Safety Council, organizations which are close to the home such as the Red Cross, parent-teacher associations, public health organizations, etc., are attacking the home accident problem. The American Public Health Association has a Committee on Home Accidents.

Child accidents are now the leading cause of death in children from 1 to 15. Three years ago, the U. S. Children's Bureau, the American Academy of Pediatrics and the Metropolitan Life Insurance Company launched a national child safety campaign. This movement has gained ground. The American Academy of Pediatrics has now a special committee which through pediatricians in the United States, Canada and the South American countries, is working with local medical societies, safety organizations, public health agencies and other community groups to reach parents, teachers, and all those who are responsible for the welfare of children, with the facts about and ways to prevent child accidents. At the present time, the Prudential Insurance Company has launched a very promising child accident prevention campaign in California. The remarkable feature of all this preventive activity is the interest and enthusiasm shown by physicians, particularly pediatricians.

#### *School and Industrial Hygiene*

We pointed out as one of the earmarks of public health its concern with the group approach. We must not overlook the access which public health has to large segments of the population through the schools and through industry. In the United States, about two thirds of all the children of school age are exposed to periodic health examinations which emphasize the discovery of adverse health conditions and their treatment or correction. This has some implications for life insurance. While many of these examinations leave much to be desired from a clinical point of view, undoubtedly many children with physical defects, which might cause their rejection or an increased rating, have had these corrected. Of greater importance, perhaps, is the increased emphasis being put on health and safety teaching through the schools. Health education which helps the child acquire proper attitudes and practises concerning nutrition, rest, relaxation, and human relationships in every day living can make an important contribution toward life expectancy.

Industrial hygiene, in many ways comparable to school hygiene, likewise has contributed to life insurance by its

concern with protection of the health of the worker. Studies of working conditions, including exposure to toxic substances in the environment, and the development of control measures have been important factors in reducing premature death and disability. Preplacement and periodic health examinations and health education programs are helping to maintain the health of workers and discover disease in its early stages.

Neither school nor industrial health programs have realized their potential. Through these channels, public health in the future may make important contributions to life insurance. Virtually untouched, for example, is the opportunity to follow groups of children and adults over a long period of time. Valuable information on prognosis could be obtained which should be useful in medical underwriting.

What, then, in brief, has public health contributed to life insurance? By reducing the number of premature deaths among wage earners, the economy has benefited and this in turn has been a contribution to the life insurance business. In addition, it has meant that a greater number of young persons in the population have been eligible for life insurance. And, not to be overlooked, this life saving has helped to keep down the cost of insurance.

#### *Future Prospects*

We stated earlier that there are some who feel that public health may already have had its greatest impact on life insurance particularly as regards medical underwriting. Certainly if all that public health has to contribute is a reduction in mortality from infectious diseases, there is something to be said for this point of view. Mortality under age 35 has become so low that insurance for amounts up to \$10,000 is being sold generally at these ages without a medical examination. However, there are many major public health activities which eventually are likely to affect our business.

An organized attack is beginning on the health problems of the population over age 40. We are also involved with this increasing segment of the population because a growing number of the insured population are in this older bracket.

Today, and to a greater extent, tomorrow, mortality from the cardiovascular diseases, cancer and other diseases characteristic of an aging population do, or will, account for the bulk of the claims.

Under the leadership of the American Medical Association, the Commission on Chronic Illness has been organized to study chronic disease, illness and disability. The Commission includes, in addition to the American Medical Association, the American Public Health Association, the American Hospital Association, the American Public Welfare Association, and the other national voluntary and official agencies concerned with the many facets of this problem. It is encouraging to note that the Commission believes that "the basic approach to chronic disease must be preventive. Otherwise, the problems created by the chronic diseases will grow larger with time, and the hope of any substantial decline in the incidence and severity will be postponed many years."

We do not know enough about the causes of such important chronic ailments as heart disease, cancer and diabetes to pinpoint a preventive attack on them at the present time. Today we must put our emphasis on earlier detection and better treatment. But even this emphasis, applied on an organized basis, after the manner of attacking tuberculosis, would prevent or postpone death and disability for many people. For example, the American Cancer Society estimates that even with no new knowledge about detection or treatment, but simply by earlier diagnosis and better treatment, a third of those who now die of cancer would be saved. The American Diabetes Association, in cooperation with medical societies and public health agencies throughout the country, has for the past several years carried out very successfully diabetes detection drives. They have helped to discover many previously unknown cases of diabetes, and have also served a valuable educational purpose. In Newton, Massachusetts, the U. S. Public Health Service is carrying out a heart disease control project to learn if heart disease mortality can be reduced by better application of the knowledge we now possess.

In Framingham, Massachusetts, site of the famous tuberculosis control demonstration, the U. S. Public Health Service has begun a 20 year study of the life history of arteriosclerotic and hypertensive cardiovascular disease in the population of that community. One of the important by-products will be knowledge of the efficiency of various diagnostic procedures in finding heart disease.

Research of all types must go on, of course, and be intensified in the area of the degenerative diseases. We heard yesterday from Dr. Francis Dieuaide of the progress being made in cardiovascular research with the assistance of the Life Insurance Medical Research Fund and other sources. Meanwhile, let us encourage the application to the greatest possible extent of all that is known about these diseases. This calls for more education, both of the profession and the public.

#### *Health Education*

Health education which embraces efforts to teach the layman how to protect his health is a powerful public health weapon. It has been of unquestioned, though quantitatively intangible, value in the eradication of the diseases previously mentioned. It promises to be of even greater importance in the years ahead. We are entering an age when, more than ever, the seeds of man's destruction lie within him. In the infectious disease era, we had to protect man from the insect, from contaminated food and water—in short, to provide him with a safe place to live. We have been able, by immunology, to increase his resistance to certain of these infectious diseases. Now man must depend more on diet, sleep, relaxation, mental poise to build up resistance to disease and to maintain his health. Here it seems he must learn to help himself through knowing *how* to live with himself and his neighbors.

Perhaps there is no subject more important in health education than nutrition. Public health programs of official and voluntary agencies are placing increasing stress on the teaching of nutrition, as evidence accumulates which shows that a substantial number of our population has inadequate diets,

less through poverty than through ignorance. Moreover, there is a close relationship between an adequate diet and good health, notably maternal and infant health. Nutrition also is linked closely to the degenerative diseases.

One of the major causes of increased rating for life insurance is obesity. From the evidence available, it appears to have an intimate association with cardiovascular disease, diabetes and other degenerative conditions. Today, public health agencies are showing great interest in a weight control program. The U. S. Public Health Service is cooperating with our company and the American Medical Association in a national educational campaign. We are featuring as one tool of public education a motion picture "Cheers for Chubby" which after a premiere at Radio City several months ago, is now being shown with the assistance of state health officers, medical societies and other health agencies in theatres all over the country. We have been impressed with the interest shown in this program by professional and lay groups and individuals. The American Diabetes Association is focusing on the overweight individual in its annual diabetes detection drive next month. The American Heart Association has urged its affiliated heart organizations throughout the country to inform the public about the dangers of overweight and the importance of weight control. Many health departments, medical societies and other health agencies are making weight control a part of their health education program. To us, this is—and apparently others agree—the most constructive activity which public health agencies can undertake at the present time as a means of preventing or minimizing the effects of cardiovascular disease.

Another subject which permeates the entire range of public health activities, indeed of life itself, is mental health. Psychiatry, long enmeshed with mental disease frequently in an irreversible stage, is beginning to be more and more concerned with the possibilities of preventive measures. To an increasing degree, physicians, nurses, social workers, educators, clergymen, all of whom work intimately with people, are being given more understanding of human relationships. The

mental hygiene movement has great potential significance for life insurance. At this point, we can only speculate on the ultimate advantages to be gained by better understanding of the emotional factors which contribute to death and disability from accidents, disease and suicide acts. But it is obvious that even the elementary knowledge we now possess concerning the influence of thoughts and feelings upon human behavior and the human body, if more generally applied, could prevent some portion of untimely death and disability.

#### *Professional Education*

In recent years, more and more health departments have been staffed with well trained health personnel. There are now eleven accredited schools of public health which turn out annually more than 800 trained health officers, statisticians, laboratory directors, health educators and sanitarians. In addition, there is an army of more than 25,000 public health nurses, exclusive of those in industry, who are literally the "foot soldiers" of preventive medicine.

Future progress in the prevention or reduction in incidence of major health problems of the future depends in large measure upon having more practicing physicians trained in preventive medicine. Life insurance would profit, too, if more medical students were taught to have the same passion for health as they now have for disease. An encouraging note is the report that the Rockefeller Foundation has just made a grant of \$15,000 to bring together all the professors of preventive medicine for a several day conference with a view to reorienting and strengthening their departments in the light of their own experience and the increasing significance of this aspect of medical education.

An advantage in focusing the attention of the medical profession on the growing importance of prevention has been the creation of a new specialty board on Preventive Medicine and Public Health. Since it was organized in 1948, more than 1,700 physicians have been certified.

*Contribution of Epidemiology*

In addition to its concern with the relationship of physical impairments to life expectancy as, for example, obesity and hypertension, medical underwriting must take into account the influence of social factors such as living conditions, occupations, health habits, etc. In the investigation of the influence of social, genetic, environmental and domestic factors on the incidence of disease and disability, epidemiology is an essential tool of public health of the greatest importance.

With the increasing control of the infectious diseases, the technic of epidemiology is being applied to other health problems such as nutritional disorders, occupational hazards, cancer, rheumatic fever, to mention a few. An example of the brilliant use of this technic is the demonstration of the relationship between German measles contracted in the first 6 or 8 weeks of pregnancy and the occurrence of congenital defects in the offspring. By the application of epidemiologic methods, it was discovered that there is an apparent association between a recent intramuscular injection and the occurrence of paralysis in an upper extremity due to poliomyelitis. The correlation is of sufficient significance to make it advisable at the present time to postpone immunization and other routine injections when poliomyelitis threatens a community. The long term study of heart disease in Framingham, Massachusetts, previously referred to, is essentially an epidemiologic study. As this method of studying disease and disability in a population grouping expands beyond the investigation of epidemics, it may be expected to have more implications for insurance underwriting. This is particularly true as it relates to morbidity experienced by life insurance companies in connection with their accident and health business.

*In Conclusion*

By reducing the number of premature deaths among wage earners in this country, public health has made an enormous contribution to life insurance. Not only has this saving of human life made more people eligible for life insurance, but by keeping down the cost, it has enabled more and more in-

dividuals to purchase insurance. An encouraging and increasing degree of cooperation and understanding of public health objectives exists among voluntary and social agencies, as well as organized medicine, as evidenced by recent campaigns against diabetes, heart disease, accidents and obesity. Public health and life insurance will continue to profit by better understanding of each other's interests and potential contributions. It seems clear that in the future public health will concern itself more and more with the chronic and so-called degenerative diseases. It is important both for life insurance and public health that the emphasis continue to be on prevention rather than treatment. Prevention is infinitely cheaper than treatment.

In the end, the man in the street, who is our policyholder, gains. He gains health protection and he gains insurance protection at the lowest possible cost.

PRESIDENT YLVISAKER — Thank you, Dr. Wheatley.

"The Contributions of Life Insurance to Public Health" will be discussed by Dr. Ronald F. Buchan, Director of Employee Health at the Prudential Insurance Company of America, Newark, New Jersey.

## THE IMPACT OF LIFE INSURANCE ON PUBLIC HEALTH

RONALD F. BUCHAN, M. D.  
*Director of Employee Health*

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As an industry, Life Insurance has a live interest in and continuously contributes to the health of the nation. Many of these activities have been or will be described in detail during these meetings. It may be helpful therefore to review briefly and summarize those aspects of health activity on the part of insurance companies which contribute to public health. Of prime importance is the Life Insurance Medical Research Fund. It would be redundant for me to explore in detail the encouraging and significant accomplishments of the Fund. The subject was covered in admirable fashion by Dr. Dieuaide during yesterday's session. Let us reiterate, however, that the numerous investigations under the auspices of the Fund have had a substantial influence on the development of knowledge related to cardiovascular disease. The pursuit of promising leads, the dissemination of developed data to our practicing physicians, and their incorporation in the teaching of our medical students, interns and residents, leads to an intelligent appreciation of the factors associated with cardiovascular diseases. We are not presumptuous when we predict a salutary effect upon the management of these cases leading to enlarged concepts of productivity consistent with cardiac reserve. I am sure the economic implications of continuing activity in these patients are apparent. We must admit, however, that with the satisfaction of maintaining economic equilibrium for many younger cases, we create the equally challenging problem of occupational and social integration of an increasing number of older cardiac patients.

Health education next attracts our attention. We are all familiar with the various media of education which have been

developed, supported, and advanced by insurance companies. The audience reached by the literature, radio, and films of the insurance fraternity extends to the youngest schoolboy. It is difficult to measure the actual influence of such educational devices upon health habits and practice. It is not unreasonable to presume, however, that the repetition of this message over the years could fail to influence the health thinking of numerous of our citizens.

More measurable, however, are such special projects as case finding directed towards diabetic and tuberculous patients. It is quite generally agreed that such measures result in the detection and consequent medical care of many thousands of cases per year. In addition, such projects as obesity control cannot fail to have an influence upon one of our most easily remediable physical impairments. The long train of associated pathology is generally too well known to this audience for me to dwell upon it, and suffice it to say that corrective measures initiated by representative members of the life insurance industry contribute to the general well-being of our whole population. Later this afternoon, you will hear much more about this problem of obesity.

A prominent insurance executive stated before this Association two years ago that if, as an industry, we were to conduct and support employee health services, our efforts should be exemplary and a model for other industry. I think the wisdom of this is apparent to all of us and there is no question but that through our existent employee health services, case finding in every category is at a high level. The number of cases returned to community practitioners and facilities for care of medical and surgical conditions is at a high level. In my own Company, for instance, we have returned to family doctors, specialists, and community facilities some 4,000 cases during the last year. This, of course, is exclusive of patients who have voluntarily sought out medical care motivated by our health activities. This latter factor does not lend itself to accurate measurement. The impact which we can have upon continuing mental and physical health through our contacts, consultation and counseling with our employees is, I am

sure you will agree, a significant factor in the continued level of healthy, happy productivity in the community. At the same time, our messages are carried into the homes of our employees. The showing of such films as "Self Examination of the Breast", are helpful not only for the employee, for we know that its message is transmitted to many friends and members of the family, contributing to the total health education effort previously mentioned. The opportunity for epidemiologic and clinical investigation with our controlled working population has not gone unrecognized. A survey of medical literature for the last five years, for instance, will reveal a solid contribution by the medical departments of insurance companies, exclusive of the activities of this Association.

In an analogous fashion we consult, upon request, with the attending physicians of rejected or rated applicants for life insurance, effecting in many instances proper medical supervision of patients who otherwise would not have been aware of their incipient difficulties.

Our laboratories have developed new technics, and have refined or improved old ones. Not only in the field of the clinical laboratory have these efforts been effective, but also in the laboratory of industrial hygiene. In the latter, the potentialities are far reaching, as the control of noxious working environments has a specific preventive effect. One need mention only the changed outlook in incidence of silicosis in foundries and quarries for example, or the control of the beryllium hazard in varied industries including our Atomic Energy Commission installations. In similar fashion, special studies in underwriting research related to cardiovascular diseases and tuberculosis, such as those described during this meeting, develop fundamental information which has not only the practical value of assisting in our daily underwriting tasks, but leads to more intelligent appraisal of situations encountered by the family physicians and specialists. The awards made at exhibitions of the American Medical Association and state medical society meetings to companies rep-

resented by our membership give tangible evidence of the value attached to these scientific undertakings and exhibitions.

A feature of growing importance in the health of our community population is the effect of hospitalization, accident, and sickness plans. This, of course, is an extensive subject in itself, and I do not intend to quote statistics. Let it be said, however, that the medical, surgical, and hospital care afforded large segments of our population through insured group and individual plans is producing definite improvement in the health and economic status of our people. Indeed, in large measure such plans are solving, one after another, major problems of health care and maintenance widely recognized as one of our most important medico-social problems.

Thus, to summarize briefly, it may be seen that our organized efforts have an influence upon the health pattern of this nation, particularly as effected through the following media:

1. The Life Insurance Medical Research Fund.
2. Health education in all its aspects—literature, radio, film.
3. Special health projects as represented by diabetes and tuberculosis detection programs and obesity control.
4. Employee health services—their case finding technics, epidemiological and clinical investigation, and their basic function of the conservation and maintenance of health.
5. The bringing under medical care of those insurance applicants who receive rejected or rated classification.
6. The development of new, and refining of old clinical laboratory diagnostic methods. The development of and preventive application of industrial hygiene laboratory technics.
7. Underwriting research with its valuable contribution of the development of basic clinical information relating to special categories such as cardiovascular disease and tuberculosis.

8. The challenge of medical care for all age groups as represented by the provisions of group accident, sickness, and health insurance policies and the wide coverage effected thereby in the employed population.

Indeed it is apparent that the insurance industry is accepting the full challenge of its share in the problems of community health. The contributions of this industry find daily application in the efforts of organized official and voluntary health agencies as well as in the responsibilities of the practicing physicians of the community. Combined with the outstanding accomplishments of our official and voluntary agencies in the field of public health, we can look forward to a continued and constantly improving health status for our people.

**PRESIDENT YLVISAKER**—Thank you, Dr. Buchan. I am sure these public health discussions will impress all of us with our responsibilities in this field.

Our program committee felt that developments in the gastrointestinal field should be brought up for review and that the relation of these developments to our underwriting problems should be discussed at this meeting. Dr. Franz J. Ingelfinger, Professor of Medicine at the Boston University School of Medicine, will open this discussion. Dr. Ingelfinger—“The Prognosis of Benign Gastrointestinal Conditions.”

## THE PROGNOSIS OF BENIGN GASTROINTESTINAL CONDITIONS

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The average patient who comes to his physician with digestive complaints is suspected of having a biliary disorder, peptic ulcer, or carcinoma. To confirm or exclude these possibilities, x-rays are ordered. The x-ray thus occupies the position of a final arbiter; according to its decision, the patient does or does not suffer from organic gastrointestinal disease. This is unfortunate because the radiologist, mindful of the responsibility placed upon him, uses all his great skill and highly developed technical facilities in a determined effort not to "miss" anything. In this effort he may discover and describe a number of conditions which are neither ulcerous nor cancerous but which appear to him as definite abnormalities in the structure or function of the gastrointestinal tract. These conditions include diaphragmatic hernia, gastritis, diverticula of the esophagus, stomach, small bowel and colon, the poorly functioning gallbladder, and—a more recent arrival—prolapsed gastric mucosa.

When the referring physician receives the radiologist's report, it is up to him to decide whether or not the abnormalities described account for the patient's symptoms. Since human nature in the persons of both doctor and patient abhors the unexplained and the mysterious, the physician is usually quite content to put two and two together and to explain his patient's symptoms on the basis of the observed x-ray defect, be it a diaphragmatic hernia, a duodenal diverticulum, a malfunctioning gallbladder, or a spastic colon. In this manner the practitioner has endowed a considerable number of radiologic abnormalities with clinical significance so that now we discuss the medical manage-

## BENIGN GASTROINTESTINAL CONDITIONS 207

ment of these disorders, we debate the necessity of surgical intervention, and we concern ourselves with the prognosis.

In your field the question of prognosis is of paramount importance. You want to know whether the patient will be incapacitated by his symptoms, whether he eventually will require surgery, or whether he will suffer fatal complications. To answer these problems in terms of means and standard deviations requires a longer period of followup and an interest by insurance companies in diverticula comparable to that manifest in hypertension. Since the day on which insurance companies will establish a richly endowed foundation for the study of prolapsed gastric mucosa or spastic colon does not appear on the immediate horizon, an estimate of the prognosis of the benign gastrointestinal disorders mentioned must be made on the basis of personal experience and the available medical literature.

Unfortunately, rough estimates of this type can be expressed only in terms of the over-all incidence. A breakdown according to age is not feasible. This is particularly unfortunate because most of the benign conditions of the gastrointestinal tract that I shall discuss are rare in youth and common in old age. On the other hand, your principal concern is probably not so much the incidence of benign disorders, many of which remain asymptomatic throughout life, but the incidence of serious and potentially fatal complications resulting therefrom. It is a reasonable guess that the likelihood of such complications in a young or middle-aged person with a benign gastrointestinal abnormality is at least equal to, or perhaps even greater than the over-all incidence of complications of this disorder at all ages. For example, a man of 35, discovered to have diverticulosis of the sigmoid, has plenty of opportunity during the remaining years of his life to develop more diverticula and their several untoward sequellae.

### *Diaphragmatic Hernia*

The lower end of the esophagus, although an area easily accessible to diagnostic tools, is surprisingly enough a sub-

ject of considerable controversy. The existence of functional and anatomic sphincters in this area is debated. It is, however, evident that the distal few inches of the esophagus may not exhibit the same peristaltic activity as the rest of the organ. Following a barium swallow, barium may puddle in this distal portion of the esophagus to form a triangular area known as the phrenic ampulla. Differentiation of a large phrenic ampulla from a diaphragmatic hernia is often extremely difficult and largely a matter of opinion. Since esophageal mucosal folds may be coarse and gastric folds thin, the appearance of the mucosal folds as the gut passes through the diaphragm is not the reliable criterion it was once believed to be. For these reasons, pockets of barium that persist above the diaphragm, particularly when the patient swallows in the supine or prone position, may be considered to be in the esophagus—and hence normal—by some observers, whereas others will interpret the picture as that of a diaphragmatic hernia. In other words, one radiologist sees diaphragmatic herniae in over 40 per cent of his elderly patients, while another makes the diagnosis relatively infrequently. My own belief is that recognition of diaphragmatic hernia as a clinical and radiologic syndrome was followed by a period of over-enthusiastic diagnoses, and that many patients who are said to have a diaphragmatic hernia have no more than a prominent phrenic ampulla. Perhaps I am overly skeptical, but I want to emphasize that the radiologic diagnosis of a small diaphragmatic hernia—i.e., one less than 5 cm. in diameter—is not easy, even in expert hands. For these reasons I believe the diagnosis of diaphragmatic hernia in an applicant for insurance should be carefully checked and evaluated, particularly if the hernia is small and the applicant has either no symptoms or very atypical complaints.

If a large hernia is demonstrated radiologically, or if symptoms characteristic of this condition are elicited, the diagnosis can be accepted with little hesitation. That diaphragmatic herniae may produce symptoms is of course unquestioned.

## BENIGN GASTROINTESTINAL CONDITIONS 209

Pain in the upper abdomen or lower chest, often radiating around the costal margins or up under the manubrium, is a common complaint. Often the patient suffers from epigastric pressure, fullness, heartburn, acid regurgitation and even vomiting, all of these symptoms being intensified by a full meal, by bending over, or lying down. Bleeding, pain simulating angina pectoris, and interference with the respiratory function are rarer phenomena. Sudden death from strangulation, perforation or exsanguinating hemorrhage may occur but is so unusual as to be relatively insignificant from the insurance standpoint.

Patients with large or symptomatic diaphragmatic herniae do, however, face the risk of an operation which may be necessary to correct pain, bleeding, or difficulty in swallowing. (1) A few cases may be relieved by the relatively safe procedure of a phrenic crush, but, in the majority, the risks of a major transthoracic operation must be undertaken. How many patients less than 50 years of age and suffering from either symptomatic or large diaphragmatic herniae will eventually require major surgery? As a rough approximation, the figure of 1 out of 20 may be used. Elderly women appear to be several times more susceptible to bleeding or incapacitatingly painful diaphragmatic herniae than elderly men, but it is not clear whether this difference indicates that women have more diaphragmatic herniae, or that they are more likely to develop complications.

In summary, it seems that the risks of insuring a patient with a diaphragmatic hernia are minimal if the hernia is small and does not produce typical symptoms. In larger herniae or those producing symptoms, the risks of major surgery in elderly and often obese subjects must be weighed.

### *Cardiospasm*

Another condition producing obstructive phenomena at the lower end of the esophagus is so-called cardiospasm or achalasia of the esophagus. In spite of its name, which implies that a local motor dysfunction at the cardia is primarily responsible for this condition, studies in our laboratory have

shown that the entire lower two thirds of the esophagus is involved in this disease. (2) In advanced cases it is a severely disabling disorder. The esophagus is dilated and tortuous, weight is maintained with difficulty, secondary pulmonary disorders may arise, and sometimes esophago-gastrostomy is performed in an attempt to improve nutrition. Many of these operations, however, are proving quite unsuccessful because the patient develops an esophagitis equally disabling as the cardiospasm for which the operation was undertaken. For this reason, a patient with cardiospasm and clear-cut esophageal dilatation must be considered a substandard risk from the viewpoint of insurability. On the other hand, patients with early cardiospasm who, following dilatation of the cardia, have lead an asymptomatic and healthy existence may be considered near-normal risks.

#### *Gastritis*

From the vast subject of gastritis, a few facts may be selected that pertain particularly to your interests. In the first place, only one type of gastritis, the so-called giant hypertrophic gastritis, can be diagnosed by an x-ray examination, and this type of gastritis is quite rare. The much more common types of gastritis, superficial, hypertrophic and atrophic, can be diagnosed only by gastroscopic observation or direct inspection of the stomach at the time of a laparotomy. Thus x-ray diagnoses of gastritis which are based upon prominence or tortuosity of the mucosal folds should not be accepted without further study. It is of course implicit in any diagnosis of pernicious anemia that the patient suffers from atrophic gastritis. Similarly, any patient who is shown to have histamine anacidity by gastric analysis probably suffers from some atrophic gastritis.

Although many patients lead perfectly normal lives in spite of the fact that they have histamine anacidity, atrophic gastritis, or both, this group as a whole appears to be more susceptible to the development of gastric neoplasm, both benign and malignant, than are patients with normal gastric mucosa. The incidence of gastric carcinoma in patients with pernicious anemia is estimated to be three to four times as

## BENIGN GASTROINTESTINAL CONDITIONS 211

high as in the population at large. (3) The diagnosis of atrophic gastritis, particularly if based on both gastroscopy and gastric secretory study, would therefore seem to decrease insurability considerably.

The risks of the other types of gastritis are less. Some forms of hypertrophic gastritis may eventually merge into the atrophic type. All types of gastritis may bleed, but the bleeding is usually an oozing rather than an acute massive hemorrhage. A few patients with giant hypertrophic gastritis have sufficient distress to require surgery. Thus, a few patients with gastritis may be exposed to the risks of a subtotal gastrectomy, but the proportion of patients with gastritis who eventually require surgery is unknown, chiefly because the incidence of gastritis itself is unknown.

### *Prolapsed Gastric Mucosa*

Within the last five years, numerous articles have appeared in the American medical literature describing the appearance and symptoms of prolapsed gastric mucosa. X-ray studies of the pyloric area show that gastric peristalsis may force a mushroom of redundant mucosal folds through the pylorus and into the base of the duodenal cap. It is postulated that this prolapsed mucosa obstructs the pylorus and produces symptoms of indigestion-epigastric fullness, pain, nausea, vomiting, belching.

Although clinical interest in this prolapsed gastric mucosa is recent, the mobility of the gastric mucosa of the stomach has been recognized for years. The submucosal tissue, particularly in the area of the antrum, appears to be very loose and permits a free sliding back and forth of the mucosa over the deeper layers of the stomach. It is perfectly normal therefore for gastric peristalsis to push a certain amount of gastric mucosa ahead of it, and squeeze a few folds through the pylorus and into the base of the duodenal cap. The question is this: Is the amount of prolapsed mucosa ever large enough, or does it ever remain long enough in the duodenal cap to produce the symptoms ascribed to it? In my opinion the answer is an unequivocal "no". We do not diagnose

prolapsed gastric mucosa as a cause of symptoms in our clinic. To do so is to seek desperately for a simple and obvious mechanical explanation for symptoms that may have a more subtle and functional basis.

#### *Diverticulosis and Diverticulitis*

As people get older, one of the signs of advancing years is the appearance of mucosal herniations through the wall of the gut, giving rise to the condition known as diverticulosis. These pouch-like projections from the gastrointestinal lumen are very common in the colon, particularly its sigmoid portion. The incidence of colonic diverticula is usually said to be about 10 per cent in patients over 45. This incidence increases sharply with age, and in our own experience colonic diverticula are found in one third of the patients more than 60 years of age. In patients less than 40, diverticula are discovered infrequently (4) (5). It should be emphasized that these figures are based upon x-ray examinations. Consequently, the figures quoted actually do not represent the incidence of diverticulosis in the population at large; rather they give the incidence of diverticulosis in people who have been given barium either by mouth or by rectum.

In other parts of the digestive tract than the colon, diverticula are less common but still not rare. A favorite site is the descending duodenum near the ampulla of Vater. The incidence of diverticula in this or adjacent portions of the duodenum is commonly quoted as about 1 per cent, but these figures, at least in our experience, are much too low. In patients having barium studies of the upper gastrointestinal tract in our clinic, duodenal diverticula appear in 4 per cent. This discrepancy again represents, at least in my opinion, the interest and attitude of the radiologist who wants to find something. If he happens to find a cancer of the stomach, he will describe this in detail and may not mention the incidental and unimportant finding of a small duodenal diverticulum. Thus, estimates of the incidence of duodenal diverticula are lower than they should be, particularly since the estimates are usually made by reviewing x-ray reports, not by reviewing x-ray films.

## BENIGN GASTROINTESTINAL CONDITIONS 213

As an example of the competing influence that two simultaneously existing abnormalities may exert on the radiologic interpretation, I should like to cite an article (6) which appeared to show that patients with diaphragmatic herniae are less susceptible to cancer of the stomach than the population at large. In a review of the reports of 3,448 gastrointestinal x-ray examinations, cancer of the stomach was diagnosed in 2.92 per cent of all patients studied and only in 0.65 per cent of the 308 patients with diaphragmatic hernia. Now, why this difference? To me, it seems likely that the radiologist, when confronted by a possible neoplasm, might easily fail to mention the associated and quite unimportant finding of a small diaphragmatic hernia.

In addition to appearing in the duodenum, diverticula may occasionally be found in the esophagus, the cardia of the stomach and the remainder of the small intestine. Unless really huge—let us say over 2" in diameter—diverticula in these parts of the gut should be considered asymptomatic and of no prognostic import. This statement is made in the face of many case reports in the literature ascribing this and that symptom to gastric or small intestinal diverticula. What happens is usually this. A patient has digestive symptoms—"gas", pain, distress, nausea, cramps, and even intestinal blood loss, and his doctor orders x-rays. If careful study reveals nothing except a duodenal diverticulum, the doctor is sorely tempted to satisfy his inner demands for security and his patient's demands for an explanation by ascribing the symptoms to the obvious defect in the duodenum. In spite of the recent emphasis on psychosomatic disorders, many doctors and certainly most patients still want some nice concrete and organic scapegoat on which all aches and pains can be blamed. In making this correlation between symptoms and x-ray findings, however, the doctor ignores the fact that in many, many patients—as a matter of fact in the majority of patients visiting any gastrointestinal clinic or specialist—no organic defect whatsoever can be found to explain the symptoms. In these instances, the doctor and the patient must perform content themselves with a diagnosis of

"nervous stomach", functional disorder, or the like. In other words, exactly the same symptom complex is ascribed in one case to a competitive anxiety state and in the other to a duodenal diverticulum, and all because the radiologist has found one little harmless pouch projecting out from the duodenal lumen.

A recent case report (7) provides a beautiful text for this particular sermon. The case is that of a 77 year old woman who suffered from repeated gastrointestinal bleeding, but x-rays revealed only diverticula of the large bowel, the duodenum and the jejunum. Because the colonic diverticula were suspected as the source of her bleeding, a colostomy was performed, but the patient bled again, obviously at some site above the cecum. At this point, the jejunal diverticula were suspected and were removed surgically although no obvious source of the bleeding in these diverticula was found. The authors then commented on this case saying, "The true significance of the presence of jejunal diverticula was not recognized at the time of the first operation. When bleeding continued postoperatively, the importance of the diverticula was appreciated. The involved segments were subsequently removed and recovery followed." An honest addendum, however, appears at the end. One year later the patient bled again, whereupon her duodenal diverticulum was resected. Three months later she was back with more intestinal bleeding and, in spite of 12 transfusions, succumbed. At autopsy no cause for the bleeding was found. Although the liver showed cirrhosis, no varices could be demonstrated.

On rare occasions diverticula outside of the colon may cause real trouble. Huge diverticula in the esophagus may produce sufficient interference with swallowing to necessitate resection of the pouches. Inflammatory diverticulitis of jejunal diverticula with perforation has been reported. These complications, however, are so unusual that the radiologic demonstration of diverticula in the esophagus, stomach, or small bowel should not impair the insurability of the patient.

In the colon the situation is different, for about one patient out of ten with colonic diverticulosis may suffer from inflam-

## BENIGN GASTROINTESTINAL CONDITIONS 215

mation of these pouches of a sufficient degree to produce symptoms. When diverticulitis occurs, it practically always affects the diverticula of the sigmoid, rarely those of the cecum. Diverticulitis may lead to serious complications—obstruction, perforation, local abscess formation, sigmoidovesical fistulae and occasionally gross bleeding. Of the patients with clinical symptoms of diverticulitis, between 10 and 20 per cent may eventually require surgery. In other words, if diverticula are demonstrated in the sigmoid by x-ray, the chances are about 2 per cent that the patient will eventually have a serious complication on the basis of this disorder or will have to undergo major surgery because of it. As a matter of fact, an increasing proportion of patients with diverticulitis may be subjected to surgery because antibiotics and improved surgical technics are making surgery safer, and medical treatment is on the whole rather ineffective in preventing recurrent attacks of diverticulitis.

One question that is often raised is whether diverticulosis or diverticulitis of the colon leads to cancer. The evidence appears to be conclusive that this is not the case. In patients suffering from both cancer and diverticulosis of the sigmoid, however, the presence of the diverticula may obscure the radiologic characteristics of the cancer and may delay its recognition.

### *Disorders of the Biliary Tract*

The x-ray provides us with our most reliable means of diagnosing disorders of the biliary passages. Two that I should like to discuss are the silent gallstone and the poorly functioning gallbladder, for these conditions represent to me the opposite extremes in terms of clinical and prognostic significance. The diagnosis of cholelithiasis by a competent radiologist is highly accurate and stands by itself. No correlation with clinical symptoms is necessary, for we know that many people have asymptomatic gallstones. The problem hotly debated in clinical circles is what proportion of silent gallstones discovered in people less than 65 years of age will become articulate and cause trouble. In one study, (8) 112 patients with initially silent gallstones were followed

for a period of 10 to 20 years; 24 of these eventually were subjected to cholecystectomy, and 3 of these died of post-operative complications. Many surgeons, however, believe that this series of cases minimizes the danger. They advocate immediate removal of the gallbladder in all patients who have silent gallstones and otherwise a reasonable life expectancy, for they insist that the dangers of a simple cholecystectomy in an uncomplicated case are much less than the dangers of emergency surgery carried out in the presence of complications such as obstructive jaundice or acute cholecystitis. As a clinching argument, they add that gallstone ileus or cancer of the gallbladder may strike at those who keep their silent stones.

We may conclude that the patient with a silent gallstone faces the risks either of immediate surgery or of later serious complications. In clinics operating with highly skilled surgical teams, the risks of an elective cholecystectomy in an uncomplicated gallstone case are small, probably no more than 1 per cent. Cholecystectomy, however, is a popular procedure, and the risks for the same operation on a country-wide basis are probably more than four times as high. If a silent gallstone is left in place, is the risk of death greater? Will the patient die either because of a direct complication of his gallbladder disease or because surgery has to be undertaken under very unfavorable conditions? In answering these questions, we must remember that many people have harmless gallstones; they are found, for example, in one third of the women dying at the age of 70 or above. The risks of not removing a silent gallstone are therefore not tremendous. If we assume that they are somewhat less than 5 per cent, the dangers of having a silent gallstone are roughly the same whether it is left in place or whether immediate cholecystectomy is undertaken.

As opposed to the x-ray diagnosis of gallstones, which is highly reliable, the diagnosis of a poorly functioning gallbladder deserves only condemnation. This diagnosis is often made following cholecystography, either because the gallbladder concentrates the dye poorly or because the viscus

empties slowly after a fat meal. In the past, some radiologists have even gone so far as to report these findings as evidences of a "pathologic gallbladder". The referring physician may then be inclined to make the diagnosis of chronic cholecystitis, particularly if the patient suffers symptoms popularly associated with gallbladder dysfunction—bloating, epigastric pressure and so-called intolerance to fats. Under such circumstances, however, this diagnosis lacks substance, for neither the symptoms nor the radiologic findings warrant the assumption that the patient has anything at all wrong with his gallbladder. In considering the insurability of any applicant, he should not be considered to have gallbladder disease unless the cholecystogram shows stones or the gallbladder fails to fill at two different attempts at cholecystography.

### *Polyps*

Patients with multiple polyposis of the gastrointestinal tract can hardly be considered candidates for insurance; the risks of malignant degeneration are far too great. One syndrome described by Dr. Harold Jeghers (9) and his associates deserves mention for, although rare, it offers the clinician one of the few opportunities he gets to diagnose small intestinal disease before the roentgenologist. Multiple polyposis of the small bowel with pigmentation of the lips and oral mucous membranes is a condition with distinct familial tendencies and is characterized clinically by recurring bouts of intestinal cramps and bleeding. In the face of such a history, blotchy brown spots around and in the mouth provide the diagnostic tip-off.

A much more serious problem is presented by what Dr. Marie Ortmayer (10) calls the solitary, non-symptomatic, non-palpable polyp of the rectum or lower sigmoid. These polyps are being found with increasing frequency because procto-sigmoidoscopy is extensively used in screening tests and cancer detection clinics. If the proctologists had their way, as a matter of fact, the proctoscope would become as much an integral part of the physician's armamentarium as

is the stethescope—or perhaps I should say as the stethescope used to be. If a polyp is discovered, the examiner must settle the following questions. Is it benign or malignant? If benign on biopsy, will it later turn malignant? If electro-coagulated down to its base, will it recur? Does its presence indicate more polyps higher up, out of the reach of the sigmoidoscope?

Strangely enough the answers to these questions are the subject of heated debate, the battlefield being strewn with fantastically contradictory figures. Atwater and Bargen (11) examined the rectum and sigmoid obtained at necropsy and by using a hand lens they found a 69 per cent incidence of polyps. Helwig (12) in a larger series and without the hand lens, found the incidence about 10 per cent in autopsy material. In other series, the incidence of polyps has been reported as even less. Obviously, the discrepancies encountered must be largely a matter of definition. In clinical medicine these same problems of definition prevail. On proctoscopic examination, small, flat mucosal elevations, no more than 1 or 2 mm. in diameter and of identical color as the surrounding mucosa, may sometimes be seen, particularly if observed in profile, sitting atop a semi-circular rectal fold. Such lesions are called polyps by some and ignored by others.

Pathologists are no more in agreement about rectal polyps than are clinicians. Estimates as to the incidence of neoplasm at the time of the initial discovery of the polyp range from 4 to 70 per cent. Suspecting that pathologic criteria might vary from one examiner to another, Dr. Ortmayer (10) sent 19 asymptomatic rectal polyps to three different pathologists for histologic diagnosis. Here is what she found.

"The stained slides were examined by three Chicago pathologists of distinction independently of each other. I am greatly indebted to them for their willingness to cooperate in this manner. Their histologic reports are challenging. Classifying on the basis of benign, suspicious (called pre-cancerous by some pathologists), and carcinomatous, all three agreed in diagnosis on only 3 of the slides made from the

biopsies. Of these 3, 1 was malignant and 2 were benign. There was total disagreement on 6 of the biopsies. Each of the 6 was called by one pathologist benign, suspicious by another, and malignant by the third pathologist. This leaves 10 polyp biopsies in which the combined opinions ended 2 to 1; 8 of these 10 were pronounced benign by two pathologists, and 2 suspicious."

In such a welter of confusion, it is obvious that no valid estimates can be given as to the likelihood of malignant degeneration in polyps initially benign. If we accept Helwig's statistics that the incidence of rectosigmoidal polyps at death in all age groups is 10 per cent, this figure may be compared with a somewhat less than 1 per cent incidence of rectal and sigmoidal carcinoma at death. Allowing for the fact that many cancers in this area are surgically extirpated and that, on the other hand, many of these cancers do not originate in benign polyps, we may estimate that possibly one rectal polyp out of ten will undergo malignant change. Even if this figure is 100 per cent off the mark, the evidence suggests that any applicant for insurance who is discovered to have a rectal polyp should have a biopsy made of this polyp and, if benign the polyp removed locally. If follow-up examination after an interval of a year shows no recurrence, a good prognosis can be given.

#### *Inflammations and Ulcers*

Up to now, we have considered conditions in which some technical procedure, principally the x-ray or the sigmoidoscope, has borne the brunt of making the diagnosis. I have emphasized that the clinical significance of many of these conditions remains to be clarified. Until this is accomplished, prognosis based upon the radiologic or sigmoidoscopic finding alone does not appear valid. This point deserves emphasis, not only in clinical medicine, where we are becoming increasingly dominated by the results of the laboratory test, but particularly in your field where actuarial tables are too easily constructed on the sagging T wave, the olive green reduction in the urine, and the not invariably black and white of the x-ray picture.

In dealing with peptic ulcer, regional enteritis and ulcerative colitis, we also rely heavily upon the x-ray and the proctoscope, but our clinical knowledge of these conditions frees us from complete subservience to technical diagnosis.

*Enteritis and Colitis*

Regional enteritis, granulomatous jejunal ileitis and non-specific ulcerative colitis are such serious diseases that very few cases could be considered insurable. Two exceptions might be made. One type of patient with ulcerative colitis appears to have the disease limited to a few inches of his rectum. If the disease remains confined to the rectum for five years with no signs of extension, and if the patient is otherwise healthy, he might be accepted as a substandard risk. A similar classification might be applied to those patients who have suffered from a stenosing terminal ileitis and who, following surgical intervention, have apparently been cured for a period of five years.

You may not be asked to pass judgment upon many cases with enteritis or ulcerative colitis, for, if the psychiatrists are right, these patients are not the type of game your agents would pursue with their usual tenacity. Psychiatric opinion is fairly unanimous that patients with these chronic, non-specific inflammations of the intestines are not of the stuff which makes for financial success in this world. This belief is epitomized by a story told several years ago by Dr. Sullivan (13) of New Orleans:

"The observations gave an example of what might have happened in 1929 on the fortieth floor of the Empire State Building, a floor occupied only by brokers and bankers who were there when the Stock Market crashed. What happens to that dozen individuals up there?

"Well, one of them is a banker, and according to his personality, he steals from old women and children, and goes to jail, because he must continue to try to be a 'big shot' and have plenty of money, and so he embezzles. Another one jumps out of the window because he cannot face life without money—and I understand you would have had to duck

## BENIGN GASTROINTESTINAL CONDITIONS 221

if you walked by there in October of 1929. A third individual turns to alcohol.

"What happens to the broker who has the 'peptic ulcer personality'? There isn't a man in this room who hasn't seen that same misfortune befall patients with peptic ulcer. What do they do? They roll up their sleeves, go to work and make another million in six months or a year, and, of course, get recurrences doing it, with hemorrhages or perforations.

"What happens on the fortieth floor of the Empire State Building to the man who has ulcerative colitis? He would never be there!"

### *Peptic Ulcer*

For several reasons I am limiting my discussion of peptic ulcer, by far the most important benign condition of the gastrointestinal tract, to a few comments. Your own experience with this very common affliction has made you well acquainted with its natural history. In addition, my former teacher, Dr. T. Grier Miller, presented a complete discussion of peptic ulcer before this same group no more than two years ago. Finally, Drs. McLellan and Pepper will discuss the effect of modern surgical procedures on the life expectancy of patients with peptic ulcer.

What Dr. Miller said about peptic ulcer two years ago is more or less true today. One might be inclined to question the 95 per cent accuracy he ascribed to roentgenologic study in the diagnosis of peptic ulcer. Perhaps this figure represents the accuracy of the superb x-ray department in his own hospital, but the radiologic diagnosis of duodenal ulcer throughout the country is probably no more than 85 per cent accurate and, in the case of gastric ulcer, is considerably less than this. It must be remembered that spot film techniques and mucosal relief studies are not practiced uniformly throughout the country. Without these, the accuracy of diagnosing peptic ulcer drops off sharply, many small ulcers being missed, and some false positive diagnoses being made.

In discussing gastric ulcers, Dr. Miller stated that ulcers in the pyloric area must be particularly suspected of neo-

plastic degeneration. This once popular view has been challenged in several quarters. It appears that any radiologically benign ulcer at or near the lesser curvature of the stomach has a 10 per cent possibility of being malignant, whether it is near the cardia, near the pylorus, or in the middle of the stomach. In my experience, this possibility is somewhat higher in men, but relatively rare in women. As Dr. Miller emphasized, however, the prognosis of a patient with a benign gastric ulcer is extremely good once the lesion has been removed surgically. Ulcer recurrences or other complications in this type of case are extremely rare.

Since Dr. Miller spoke, Banthine® has swept across the therapeutic horizon, now closely pursued by a host of imitative anticholinergic agents. Banthine has doubtless contributed to our medical care of the ulcer patient; but will it prevent recurrences, will it decrease the incidence of fatal hemorrhages, and will it decrease the number of operations necessary for intractability, obstruction, and perforation? No. It appears that Banthine will decrease the incidence of these threats to the ulcer patient's life but little. The potentialities of Banthine in reducing the complications of ulcer are limited by two factors. In the first place, not every one can tolerate this agent; in fact it is beneficial and well tolerated by only about half of the ulcer patients to whom it is given. Secondly, human nature being what it is, most ulcer patients will not take medicine regularly year in and year out, even Banthine.

If peptic ulcer is a psychogenic disorder, and since psychotherapy has become increasingly available and skilled during the past decade, one might expect that the incidence of this disorder should be declining. If such a decrease has occurred, it is so small as to have escaped detection. Whatever the reason, it must be concluded that the emphasis on the psychogenic aspects of the acid-peptic disorders has not altered the insurability risks of your peptic ulcer patient.

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## BENIGN GASTROINTESTINAL CONDITIONS 223

After all, the life situations of an ulcer patient may be so complex as to baffle not only the internist and surgeon but also the psychiatrist.

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PRESIDENT YLVISAKER — Thank you, Dr. Ingelfinger.

Dr. Lawrence L. McLellan and Dr. D. Sergeant Pepper of the Provident Mutual Life Insurance Company of Philadelphia will continue the discussion of our gastrointestinal underwriting problems. Their paper, "Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers," will be presented by Dr. McLellan.

VAGOTOMY AND SUBTOTAL GASTRECTOMY:  
EFFECT ON INSURABILITY OF INDIVIDUALS  
WITH GASTRIC AND DUODENAL ULCERS

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*and*

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It is a difficult assignment to follow Doctor Ingelfinger with his excellent over-all clinical picture of gastroenterologic conditions. Consequently my remarks will be confined to a small part of the subject of peptic ulcer. The presentation will be brief.

Three years ago Dr. McLeod Wilson (1) showed us that 10 per cent of the population have either the history or the findings suggestive of peptic ulcer, and since most persons having an ulcer have recurring attacks, we will frequently meet the problem in our applicants. He also showed that at least 75 per cent of persons with ulcer live out their lives with only a minimum of trouble, and that only 10-15 per cent of persons with ulcer ever get into serious difficulty. The mortality was shown to be inconsequential and in the same category as the automobile and aviation passenger hazard.

This paper is concerned with the 10-15 per cent of persons who get into serious difficulty, usually requiring surgical treatment. We exclude the aged and those with serious physical impairments.

We will go back a little historically to get our bearings. In the 1920's one frequently heard the advice, "Why bother with complicated ulcer management, why not just have a new opening made in your stomach and go on your way?" Fortunately some clinicians stood out against gastroenterostomy, saying that it was an unphysiologic procedure which should

## VAGOTOMY, SUBTOTAL GASTRECTOMY: 225

be used only when an ulcer had burned itself out, leaving symptoms due entirely to obstruction. In the 1930's, however, the experiments on Mann-Williamson dogs, plus favorable reports from Europe, showed that subtotal resection of the stomach was a superior operation for the severe cases of active peptic ulcer that did not react well to medical treatment. Subtotal resection was less frequently followed by jejunal ulcer than had been the experience with gastroenterostomy. Since the early 1930's, subtotal resection has been performed on such a large group of cases, by so many surgeons, that we can accurately appraise the results.

A typical experience was reported in 1951 by Glenn (2) in a review of several hundred cases treated by subtotal resection from 1938 to 1949. He found that there was a 98.7 per cent chance of surviving the operation, with an 87 per cent chance of remaining symptom free for at least 5 years. The mortality in several series reviewed by Palmer (3) varied from 0.9 to 8.9 per cent with an average of 3.9 per cent. In 1943 Dragstedt (4) first reported a favorable experience with vagotomy. Feeling that he could improve on the results previously reported, he began a careful study of this operation. It is too soon correctly to appraise the value of vagotomy, but we may be able to see where it gives the most promise of fitting itself into surgical procedures. We will discuss vagotomy along with other operations in the treatment of ulcer.

We will first review the problem of gastric ulcer. From an insurance point of view, which of necessity is conservative, we must consider a person with active ulcer of the stomach as having a potentially malignant condition, and evaluate accordingly. We should do this, even though the clinician decides that it is safe to give his patient medical management rather than subject him to the hazards of a gastric resection. We can probably give more favorable consideration to cases that have had a resection rather than medical treatment. We have the advantage of a pathological report. The site for a possible future carcinoma has been removed and we know that jejunal ulcer occurs much less frequently after subtotal re-

section for gastric than for duodenal ulcer (3). Vagotomy is seldom performed for gastric ulcer except possibly for those near the esophagus, since a resection for the reasons just given is much to be preferred, and it seems unnecessary to do a vagotomy plus the resection.

When a gastric ulcer is present in the prepyloric area in association with an active duodenal ulcer, it can be assumed the gastric ulcer is nonmalignant. Therefore it may be considered in the same manner as a duodenal ulcer.

We will now discuss the insurability of duodenal ulcer requiring surgical treatment. These are the cases which have recurrences in spite of good medical treatment. They have complications such as hemorrhage, obstruction, penetration of bowel wall, intractable pain, jejunal or marginal ulceration. Acute massive hemorrhage or acute perforation usually receives emergency surgery rather than a definitive operation. They will not be considered here.

What is meant by an adequate subtotal resection? A satisfactory operation is essential for a favorable result. Stewart (5) recently reviewed the criteria. First, there should be the removal of at least two thirds of the stomach. Second, all the gastric mucosa at the pyloric outlet should be removed. Third, the ulcer should be excised if possible. Fourth, the jejunal loop should be as short as feasible. Finally, the gastric and duodenal stumps should be well mobilized. Of course, technics vary from surgeon to surgeon, but it is well to review surgical reports, to be certain that an adequate operation has been performed. The mortality for a subtotal resection, which originally was as high as 16 per cent, has been gradually reduced as mentioned above, to as low as 0.9 per cent. This is the immediate operative mortality, and while no clinician wishes to expose his patient to even this risk, subtotal resection has become the operation of choice of many surgeons.

A typical finding is that of Gray and Williams (6) who studied the results of 532 cases of duodenal ulcer subjected to subtotal resection. They found that resection, plus the

## VAGOTOMY, SUBTOTAL GASTRECTOMY: 227

excision of the ulcer, gave complete relief in 83 per cent of the cases, partial relief in 13 per cent, while 4 per cent were made worse. When a resection was done but the ulcer not excised, they reported complete relief from symptoms in from 77 to 85 per cent of cases, depending upon the exact type of the operation. It appeared that the so-called anterior Polya operation gave a better result than the posterior. With posterior gastroenterostomy alone, complete relief was obtained in only 72 per cent of the cases while a pyloroplasty gave relief in only 30 per cent. Other competent observers have had similar experiences under well controlled conditions, allowing us to assume that only about 15 out of every hundred who require this operation will have further trouble.

We now come to vagotomy for duodenal ulcer. Historically it was conceived in the late 1920's, physiologic studies were made; it was tried clinically but found to be unsatisfactory. In 1943 Dragstedt (4) published his results in two cases and in 1946-47, reported 54 cases (7). The simplicity of the operation appealed to many surgeons. Since the early results of the operation are usually very favorable, it gained acceptance throughout the country. Unfortunately, it is very difficult to compare the results of vagotomy alone with the results of subtotal resection because it is usually necessary to combine vagotomy with other operations in order to obtain satisfactory results. Comparisons are made still more difficult because there is no agreement on the type of operation that should be performed in connection with vagotomy. How then are we to evaluate risks that have been vagotomized or which have had a vagotomy plus another gastric operation? Is vagotomy an accessory operation or a definitive one? If it is to be considered as an accessory one, how does it influence, or how is it affected by the definitive or secondary operation? We can best evaluate our problem by understanding some of the physiologic and pathologic effects of vagotomy.

Section of the vagus nerves to the stomach cuts one of the most important neurological pathways between the higher centers of the brain and the stomach, which influences the

secretion and motility of the stomach. Cutting this pathway causes an immediate decrease in the gastric juice secretion. Since peptic ulcer is a disease of persons frequently described as being under nervous tension, the theoretical value of this operation can be readily appreciated. A person with susceptibility to ulcer formation might have no trouble as long as his stomach is secreting juice at a normal or a low rate. But, if cephalogenic impulses through the vagus reach the stomach, increasing the volume and degree of gastric acidity to a high level, he may develop an ulcer. Section of the vagus nerve should restore the *status quo* and allow the ulcer to heal. Clinically this usually happens, so that the majority of patients obtain relief from ulcer symptoms soon after vagotomy. The symptoms of an acute peptic ulcer can be relieved by a vagotomy; however, it is generally agreed that simple ulcer should be treated not surgically but medically. Therefore an operation designed for the cure of a peptic ulcer must not only cure the ulcer but it must also cure the complications of ulcer. Here we experience trouble with vagotomy, for the following reasons. The vagus nerve to the stomach not only affects the rate of gastric juice secretion but it also is a stimulator of gastric peristalsis, so that when the nerve is sectioned the stomach becomes atonic. For the first few days after the operation, the stomach is widely dilated, but soon some of the tonicity of the stomach returns. But the end result is a decrease in peristalsis with a decrease in the emptying time of the stomach. As a secondary effect following section of the vagus, there is also an increased tonicity of the pyloric sphincter through the unopposed action of the sympathetic nerves. If there is also present pyloric obstruction due to an ulcer, there may be a serious interference with the emptying of the stomach. It was early appreciated that many failures of simple vagotomy were due to retention of food in the stomach because of the decreased peristalsis, plus any additional obstruction due to an ulcer. Dragstedt recommends that a gastroenterostomy be performed with vagotomy. Crile, Jones and Davis (8) recommend vagotomy and speak as favorably for pyloroplasty as for gastroenterostomy, yet they also mention resection. Beattie (9) in England, recom-

## VAGOTOMY, SUBTOTAL GASTRECTOMY: 229

mends vagotomy plus partial pylorectomy. Most of our surgeons follow Dragstedt's suggestion of gastroenterostomy.

Immediately we ask why we will not experience the same trouble with vagotomy plus gastroenterostomy as we used to have with gastroenterostomy. The proponents of vagotomy say that since there is no hypersecretion of acid, there will be none of the old complications of gastroenterostomy, but laboratory and clinical studies (10) suggest that the effect of vagus nerve section may last a few months or a few years. We must therefore consider that the good results of vagotomy may be only temporary. If secondary operations are performed, the patient may experience some of the ill effects of all such operations. If the final results of vagotomy are dependent on the type of operation performed with it, theoretically it would seem advisable to perform that operation which would have the least complications. This appears to be a subtotal resection. The question then arises: Would a person with a subtotal resection be a better or a poorer risk than a person who has had a subtotal resection and a vagotomy? If the good effects of vagotomy are permanent, he might be a better risk but the permanency is questioned. There is, however, one place where vagotomy seems to be of considerable value. That is where jejunal ulcer develops following a subtotal resection. These, of course, are among the most difficult cases to treat, but it has been found that vagotomy will, in many instances, give a satisfactory relief to the symptoms. We should briefly mention some of the reports on experience with vagotomy combined with other operations. Dragstedt (11) reports that of 509 patients operated between 1943 and 1950, he has found 80 per cent to be entirely well and back at their usual occupation on unrestricted diets. Walters and Golding (12) said they obtained excellent results in 81.4 per cent of patients followed one to four years, but later Walters and Fahey (13) reduced this to 70.5 per cent. A special committee of the American Gastroenterologic Society (3) conducted a nationwide survey by the questionnaire method; received results of over 2,500 operations; found that 98 per cent reported satisfaction with the

treatment. It is generally agreed that this last figure is much too optimistic, and that probably 80 per cent is the best that can be expected.

In summary, we should continue to have a favorable experience with persons with an ulcer history whom we insure. Some whom we insure after their first attack will have recurrences, but they will still be insurable, even though they wind up in the 10-15 per cent group who do not respond to ordinary medical treatment. If they become surgical cases, the risk of operation is being constantly lowered, the operative procedures are being made as physiologic as possible and designed to remove only the factors tending to prevent healing.

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## VAGOTOMY, SUBTOTAL GASTRECTOMY: 231

**PRESIDENT YLVISAKER** — Dr. Pepper will continue the discussion. Dr. Pepper.

**DR. PEPPER** — I first want to compliment Dr. Ingelfinger on his excellent paper on the Prognosis of Benign Gastro-intestinal Conditions and Dr. McLellan for his fine presentation of the surgical approach to peptic ulcer. Dr. McLellan's reference to Dr. Wilson's estimate that 10 per cent of the population suffer from peptic ulcer at one time or another certainly points out the magnitude of this problem. When we consider the number of x-rays made on this group as well as those for all the other conditions described by Dr. Ingelfinger it is easily seen what a fertile field exists for the roentgenologist. We must certainly be on our guard against over-enthusiastic or part-time x-ray men. All too frequently, also, as Dr. Ingelfinger has pointed out, the local doctor is quite content to explain the patient's symptoms on the reported x-ray findings.

When Dr. Ylvisaker first approached me concerning this program and asked me to discuss the two papers which you have just heard, he told me that they would be concerned mainly with the clinical side of the story. I decided that I would try to obtain and present the experience of a number of the larger life insurance companies as represented by their handling of these cases. Because of the recent innovations in surgical procedures, I thought that I would limit my remarks to the insurability of ulcer patients who have come to surgery.

I am deeply grateful to the medical directors of the various companies I wrote to for sending me their thoughts on the insurability of this group of applicants. Although I received a great amount of interesting data, the time allotted to me will permit me to discuss only briefly in general terms the handling of these difficult problems. I will discuss only duodenal ulcer.

My analysis of the various ratings applied by different companies for these impairments shows that the majority follow closely the best medical thinking. Although some of the

correlation may be dictated by the cold figures from the mortality tables, I am inclined to believe that it is rather a tribute to the excellent and up-to-date medical underwriting now in practice.

Vagotomy alone or in combination with other operations has not been performed for a long enough period or on a sufficiently large group to provide significant statistics. The ratings, however, reflect the present clinical opinion that at once places anyone who has had vagotomy in the group of medical treatment failures, and at the same time suggests that this operation should not be rated as favorably as subtotal gastrectomy.

The skeptics will probably claim that we fell on this happy solution only through our natural conservativeness.

The combination of vagotomy and other operations likewise is too recent to allow statistical evaluation of the subsequent mortality figures. Here the suggested ratings either closely follow those for the definitive type of operation or they are higher. The higher ratings probably follow logically on the assumption that any applicant who has had to resort to two operations naturally has had a particularly stormy course. However, it is interesting to note that of 19 companies reporting, only four had higher ratings for vagotomy plus subtotal gastrectomy than for subtotal gastrectomy alone, while of 17 reporting on vagotomy plus gastroenterostomy, the ratings of only six were the same as for subtotal gastrectomy alone and 11 were higher. This certainly suggests that Dragstedt's enthusiasm for this combination of operations is not unanimously shared by medical directors.

In general, the most favorable ratings are given for subtotal gastrectomy. Only slightly higher on the scale are those for the combination of vagotomy and subtotal gastrectomy; then still higher but grouped together in order come gastroenterostomy alone, vagotomy alone, and the combination of vagotomy and gastroenterostomy.

## VAGOTOMY, SUBTOTAL GASTRECTOMY: 233

Because I think it is interesting to see how a new surgical approach to ulcer is handled by the insurance business, I shall briefly describe the ratings for vagotomy as reported by 23 companies that retain ulcer cases subjected to subtotal gastrectomy. For comparison I will conclude with their ratings for subtotal gastrectomy, as I think this is the operation which we will probably be most concerned with in the future.

The ratings for vagotomy alone range from straight declination to only \$3.00 extra per thousand for the first year following operation. Between these two extremes, there are several other plans which, considered as a whole, make a rather bizarre picture. Of course, the impairment is regarded as less severe with the elapse of time and depending on operation results or possible complications.

The ratings for subtotal gastrectomy reflect the greater experience that life insurance companies have had with this type of operation. This large experience has made possible a more exact approximation of the necessary extra premiums needed to cover the mortality. As a result, the ratings do not vary as much as those for vagotomy. In general, this operation has a more favorable outlook as regards longevity than does vagotomy in the light of present experience.

PRESIDENT YLVISAKER — I want to thank Dr. Ingelfinger, Dr. McLellan and Dr. Pepper for their helpful discussions of our most difficult gastrointestinal problems.

Our final afternoon program is devoted to a discussion of our underwriting problems related to overweight.

In our company, over forty per cent of the insurance issued at substandard rates is due to overweight and its complications or associated impairments.

At the meeting of the American Medical Association at Atlantic City in June, one of the most significant scientific exhibits was on the subject of overweight. The exhibit was prepared by the Metropolitan Life Insurance Company in cooperation with the American Medical Association, the

Public Health Service, the American Dietetic Association, the American Diabetic Association and the Harvard School of Public Health.

The exhibit was impressive and did much to bring before our medical profession and the American public, the unfavorable significance of overweight both from a health and mortality standpoint.

Dr. Louis I. Dublin of the Metropolitan Life Insurance Company has already appeared before us this week. He gave us a wonderful talk during our Board of Life Insurance Medicine course, and will now present the results of recent Metropolitan mortality studies on overweights. Dr. Dublin.

## MORTALITY AMONG INSURED OVERWEIGHTS IN RECENT YEARS

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### I. *Introduction*

We at the Metropolitan have long been interested in the problem of overweight, not only because of its impact on life insurance but also because of its effect on the public health. We shall in the course of this paper discuss both phases, but in the main, the insurance aspects.

It is a far cry from the time when the hale and hearty stout man was thought to be a prime candidate for life insurance. That day passed when life insurance physicians and actuaries put their heads together and began to put the selection of life insurance risks on a scientific basis. Ever since, the mortality of overweights has received attention from insurance men and has been discussed periodically at the meetings of the Association of Life Insurance Medical Directors. We shall mention only certain studies briefly. The first full dress paper on build and mortality before the Association was given by the late Dr. Oscar H. Rogers (1) at the twelfth annual meeting held in the spring of 1901, just a little over 50 years ago. This paper included a report on 1,553 men who were 30 per cent or more overweight according to the standards he used. The mortality of this group was in the aggregate 135 per cent of the expected. It is interesting to note, in passing, that in his preliminary remarks Dr. Rogers said, "A certain amount of overweight has been looked upon with favor, our tendency being to consider a certain degree of hyper-nutrition desirable."

The Specialized Mortality Investigation (2), made shortly after the turn of the century, included some groups of overweights among the various classes of risks studied, but these were isolated groups. Moreover, the mortality table used as a yardstick in the study was faulty in some respects. Consequently, while the findings were of some value they did not provide accurate information on the mortality associated with overweight. They did indicate, however, that the mortality among overweights was higher than average, that it increased with degree of overweight and apparently also with a history of poor parental longevity. Overweights with abdominal girth measurements exceeding expanded chest girth also were subject to increased mortality.

Our present systems of rating overweights stem primarily from the results of study of build in relation to mortality of men, which was part of the Medico-Actuarial Mortality Investigation of 1909-1912 (3), a landmark in the scientific study of various classes of risks. That investigation, which was based upon standard risks, brought out clearly that the mortality of overweights was excessive, particularly at the older ages at issue and with increasing duration of insurance. The mortality results showed also that to some degree overweights had been generously treated insurance-wise. The findings of this investigation were soon reflected in the revision of ratings for overweights by life insurance companies.

Certain defects were recognized in the crude results of the Medico-Actuarial Investigation of Build, and consequently, the Joint Committee of the Association of Life Insurance Medical Directors and the Actuarial Society of America, which was responsible for the investigation, continued to study the matter further, and in 1918 brought out the report on "Standard Mortality Ratios Incident to Variations in Height and Weight among Men" (4). This showed by percentage and absolute departures from average weight the mortality ratios of build groups for short, medium height, and tall men according to age at issue.

In order to render easier the application of these tables, we at the Metropolitan subsequently constructed tables of

limits of overweight and underweight corresponding to various mortality ratios. The tables gave such weight figures at each inch of height according to age for mortality ratios at various intervals from 100 per cent to 160 per cent. These were presented by the late Dr. Knight (5) at the thirty-third annual meeting of the Association in November 1922. They set the pattern for our build ratings ever since, although these have been extended and revised from time to time. We have liberalized our underweight limits and tightened up on those for overweight.

The next important intercompany study of mortality according to build was made in connection with the Medical Impairment Study of 1929 (6), and included the experience on substandard risks as well as standard risks. This experience covered issues, on a sample basis at most ages, for the years 1885 to 1927, but the experience was limited to years of exposure from 1909 to 1928. The results were published in 1931. Without going into detail, it may be noted that they were generally in accord with those of the earlier study except that the mortality among persons markedly overweight was relatively more favorable. This was believed to be due to more careful selection following the Medico-Actuarial Mortality Investigation.

Since 1931, the only new data which have appeared on the subject of build and mortality are the experience on women insured in the Metropolitan (7, 8), reported at the meetings of this Association in 1937 and 1938, the experience of the Provident Mutual on male standard risks, reported by Blair and Haines (9) at the meeting of the Actuarial Society of America in May 1949, and the London Life experience on men, reported by Donald McCormick (10) at the November 1949 meeting of the Society. The Metropolitan Life study on women covering the period 1922 to 1936 took in the entire range of build. The experience on standard and substandard cases was analyzed separately. In both parts of this study, the mortality among overweight women, and especially among the obese was generally above average, although the figures did not show a consistent progression of mortality with in-

creasing percentage of overweight. The trend, however, was unmistakable. There was some indication in the findings that overweight is relatively less harmful to women than to men in terms of its effect on longevity.

Both the Provident Mutual and London Life studies also covered the whole range of weight groups and, consequently, their data on the more obese are relatively limited in extent. However, with certain exceptions in detail, both studies showed overweights to have mortality above the average, the excess increasing with the duration of insurance. In addition, these studies gave some indication that relatively the mortality, even of moderate overweights, appeared to be greater than in previous studies.

For a great variety of reasons, a new study of overweights is timely. The drastic change in the health picture in the last two decades, and, particularly, the accelerated rate of decline in the mortality from infectious diseases, especially tuberculosis and pneumonia, appeared to favor underweights among whom these conditions formerly had exacted a higher toll than among overweights. While the effects of these trends on the relative mortality of overweights were surmised by everyone, they required evaluation. We were spurred to action by the Weight Control Program which the Health and Welfare Division of our Company was preparing to launch in cooperation with the United States Public Health Service and the American Medical Association. We felt that it would be desirable to have up-to-date facts on mortality of overweights to meet any objections that the facts we quoted were based on old material. We undertook our study then with that immediate objective, but with an eye, of course, to its use in underwriting. We would have liked to broaden the scope of our study and make a general study of build and mortality, but limitations of time compelled us to confine ourselves to substandard overweights.

## II. *Materials of the Present Study*

In planning the new study, we sought to obtain an experience large enough to yield significant ratios of actual to

expected mortality when subdivided according to age, and to provide satisfactory data on the important causes of death in order to show what the situation was in this regard among overweights under recent conditions. We found that the experience on substandard issues insured between the years 1925 to 1934 and brought down to date would be generally adequate for the purpose. Our study is based on policies on white lives between ages 20 to 64 at issue, insured in our substandard branches during 1925 to 1934 and traced to the policy anniversary in 1950. We have excluded cases which were ratable either for other impairments, adverse medical history, or occupational hazards. This experience covers 25,998 men and 24,901 women. The deaths recorded down to the policy anniversary in 1950 numbered 3,713 among the men and 2,687 among the women. We have studied our cases according to age at issue, duration of insurance, the rating class to which they belonged, and according to degree of overweight. In addition, we have singled out for special analysis the experience on those risks who, subsequent to issue, were granted insurance at lower rates of premium, either as standard risks or as substandard in a lower premium class because weight reduction brought them within the weight limits of a lower premium class. We have, throughout, studied separately the experience on men and women. In analyzing the experience according to degree of overweight, we have further subdivided our material according to three broad height groups, as follows:

	Male	Female
Short	5' 0" — 5' 6"	4' 8" — 5' 2"
Medium	5' 7" — 5' 10"	5' 3" — 5' 6"
Tall	5' 11" — 6' 4"	5' 7" — 6' 0"

As we indicated earlier, our build ratings, on the basis of which these cases were limited to substandard insurance, are adapted primarily from the 1918 report of the Joint Committee of the Association of Life Insurance Medical Directors and the Actuarial Society of America on "Standard Mortality Ratios Incident to Variations in Height and Weight among

Men." The build ratings in use by the company during the period when these risks were written made no differentiation by age except for heights over 5 feet 9 inches. Limits for women were the same as for men at ages 50 and over, but were somewhat lower at ages 36 to 49, corresponding to the male limits for one inch less, and lower still at ages under 36, corresponding to the male limits for two inches less. On the basis of these standards the degree of overweight permissible within the limits of standard insurance was somewhat greater for women than for men, for younger persons than for older persons, and to some degree for persons under 5 feet 10 inches than for taller persons.

We have evaluated our results on the basis of the contemporaneous mortality experience by age at issue and duration among persons accepted for standard insurance. The same mortality table was used for men and women, but the figures for women were adjusted on the basis of the ratios of women to total mortality by age and duration experienced during this period. It should be noted here that these ratios are approximations during the later years of the experience because for various reasons the mortality data on women in our Ordinary Department were not available in the requisite detail. In evaluating the mortality by cause, we had to devise suitable measures, especially for the later years, because we did not have the data by age and duration on standard risks over this period in the same detail as we had for the death rate from all causes. The available data on the Standard experience were by attained age groups up to 74 years and the comparisons by cause in this paper are made on the basis of attained ages 25-74 years.

### III. *Findings of the Present Study*

Table 1 presents the aggregate results for men according to age at issue and by duration of insurance. Altogether, on the 25,998 cases, there were 285,224 years of life exposed to risk and 3,713 deaths. The mortality in the aggregate was 150 per cent of the expected. The mortality ratios are highest at the youngest ages at issue and decrease rather regularly

## MORTALITY, INSURED OVERWEIGHTS 241

with age at issue from a maximum of 180 per cent at ages 20 to 29 to 131 per cent at ages 50 to 64.

Table 1

### MORTALITY OF MEN RATED FOR OVERWEIGHT TOTAL CASES, BY AGE GROUPS AT ISSUE AND BY DURATION OF INSURANCE

Ratio of Actual to Expected Deaths by Contemporaneous Mortality  
Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues  
of 1925 to 1934, Traced to Policy Anniversary in 1950

Age at Issue	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths			
			All Years	First Five Years	6-15 Years	16-25 Years
All Ages, 20-64	285,224	3,713	150	133	152	160
20-29	53,679	224	180	129	189	214
30-39	107,533	912	169	165	158	185
40-49	90,915	1,539	152	134	157	155
50-64	33,097	1,038	131	112	136	135

The experience by duration of insurance has been divided into three broad classes, namely, first five years, 6 to 15 years, and 16 to 25 years after issue. At all ages combined, the mortality ratio is lowest in the first five years and highest in the longest duration group. This tendency exists regardless of age, although not with entire consistency. However, in every age group the ratios at the longest durations are higher than those in the first five years. The tendency is most marked at the youngest ages. In fact, the mortality ratio at ages 20 to 29 at issue in the first five years was only 129 per cent as compared with 214 per cent at duration 16 to 25 years.

Table 2 presents the aggregate results for women according to age at issue and by duration of insurance. For the 24,901 cases included, there were 289,412 years of life exposed and 2,687 deaths. The mortality ratio was in the aggregate 147 per cent of the expected. Unlike the situation among men, there is no definite trend in the mortality ratios according to the age at issue among these women rated because of over-

weight. The range of variation for all years of issue combined is relatively small and the mortality ratio at ages 20 to 29 is the lowest by a small margin. In part, at least, the narrow range of the ratios may reflect the age differences in the weight limits, as outlined above. These were more generous for older women than younger women. The mortality ratios in the three broad duration groups vary little, except at the youngest ages, where the mortality is lowest in the first five years and increases significantly at the longest durations. These differences between the sexes in the characteristics of overweights by age and duration are noteworthy and we believe them to be genuine. Our actual numerical results may be affected by the necessity of estimating mortality among standard female risks at later durations, but any errors in the assumptions we made would not be sufficient in themselves to account for these sex differences in overweight mortality.

Table 2

MORTALITY OF WOMEN RATED FOR OVERWEIGHT  
TOTAL CASES, BY AGE GROUPS AT ISSUE AND BY  
DURATION OF INSURANCE

Ratio of Actual to Expected Deaths by Contemporaneous Mortality  
Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues  
of 1925 to 1934, Traced to Policy Anniversary in 1950

Age at Issue	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths			
			All Years	First Five Years	6-15 Years	16-25 Years
All Ages, 20-64	289,412	2,687	147	151	144	149
20-29	51,594	143	134	118	134	151
30-39	113,694	703	152	153	149	157
40-49	99,696	1,225	150	168	142	154
50-64	24,428	616	138	128	146	132

*Results by Rating Classes*

In this part of our analysis, we have divided our material into two broad groups, those given an Intermediate rating

## MORTALITY, INSURED OVERWEIGHTS 243

and those given Special Class or Special Class B ratings, the latter being the highest rating group in this company up to recent years. Our Intermediate classification during 1925-1934 covered classes with expected mortalities ranging from 30 to 50 per cent above standard; Special Class, from that point up to 100 per cent above standard, and Special Class B, from there to 150 per cent above standard. About three fourths of the substandard overweights in this experience were in the Intermediate group. Both Special Class groups have been combined because the number limited to Special Class B was relatively small.

Table 3 presents the facts for men according to the two broad rating classes. In the aggregate, the mortality ratio for men classified as Intermediate was 142 per cent, as compared with 179 per cent for those insured as Special Class risks, a difference of 37 percentage points. Thus, in the

Table 3

### MORTALITY OF MEN RATED FOR OVERWEIGHT EXPERIENCE ON CASES RATED AS INTERMEDIATE AND SPECIAL CLASS SEPARATELY, BY AGE GROUPS AT ISSUE AND DURATION OF INSURANCE

Ratio of Actual to Expected Deaths by Contemporaneous Mortality Experience on Standard Risks  
 Metropolitan Life Insurance Company, Ordinary Department, Issues of 1925 to 1934, Traced to Policy Anniversary in 1950

Age at Issue; Duration	INTERMEDIATE			SPECIAL CLASS AND SPECIAL CLASS B		
	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths
All Ages, 20-64	215,052	2,715	142	70,172	998	179
20-29	39,969	152	163	13,710	72	231
30-39	80,301	620	152	27,232	292	220
40-49	68,651	1,126	146	22,264	413	172
50-64	26,131	817	128	6,966	221	143
1-5 Years	76,560	458	125	26,073	180	155
6-15 Years	102,791	1,367	144	33,608	507	178
16-25 Years	35,701	890	150	10,491	311	196

aggregate, the mortality of all Special Class risks was about one fourth higher than that of the Intermediate cases. The mortality ratios for the combined Special Class cases are consistently above those of Intermediate cases in each age group at issue and in each of the broad classes by duration of insurance. The margin of difference is much larger under age 40 than over that age. Also, in each of these two broad rating classes separately, the mortality ratios show the same general trends by age and by duration of insurance that were found in the aggregate.

Table 4 presents the comparable data for women. For them, too, the combined Special Class risks have consistently the higher mortality ratios by age groups at issue and by duration. The differences tend to be somewhat smaller than

Table 4

MORTALITY OF WOMEN RATED FOR OVERWEIGHT  
EXPERIENCE ON CASES RATED AS INTERMEDIATE  
AND SPECIAL CLASS SEPARATELY, BY AGE GROUPS  
AT ISSUE AND DURATION OF INSURANCE

Ratio of Actual to Expected Deaths by Contemporaneous Mortality  
Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues  
of 1925 to 1934, Traced to Policy Anniversary in 1950

Age at Issue; Duration	INTERMEDIATE			SPECIAL CLASS AND SPECIAL CLASS B		
	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths
All Ages, 20-64	212,167	1,927	142	77,245	760	161
20-29	38,522	102	127	13,072	41	154
30-39	83,093	488	144	30,601	215	174
40-49	71,926	865	146	27,770	360	162
50-64	18,626	472	136	5,802	144	147
1-5 Years	73,036	365	141	27,815	169	175
6-15 Years	103,391	947	141	37,440	354	151
16-25 Years	35,740	615	142	11,990	237	169

for men, but that may reflect a smaller margin in the average degree of overweight between the two broad rating classes among women as compared with men.

#### *Results by Degree of Departure from Average Weight*

While this division of the material parallels to some degree the division according to rating class, the two are not identical because, as pointed out earlier, the weight limits for standard insurance are relatively more generous for women than for men, for younger persons than older persons and for average and short persons than for taller persons. We have, therefore, divided our material on the basis of the per cent departure from average weight for height and age and have analyzed our experience on this basis of the substandard cases combined as well as in the two broad rating groups. Because of the differential according to height in build ratings, we have further analyzed the results in three broad height groups, as previously defined. We shall present details only for the combined experience on all substandard branches, but shall make reference to the experience in the separate branches in discussing the results.

The grouping by weight classes starts with those under 30 per cent overweight. Virtually all of these are between 20 and 30 per cent overweight. From that point, we have used 10 per cent classes from 30 up to 60 per cent overweight, and a broad class, 60 to 74 per cent overweight. There were some cases in the experience with greater departures from average weight, but this group, as well as some of the others, was too small to yield reliable mortality ratios.

Taking the experience on men first (Table 5), we found that at all ages and at all heights combined there is a fairly steady upward progression of the mortality ratios with increasing departure from average weight. Thus, the mortality rises from approximately 1½ times the expected for the groups up to 40 per cent overweight, to nearly 3 times the expected for those 60 to 74 per cent overweight. The increase in mortality with degree of overweight is found also in each of the height groups. The mortality ratios are highest

## SIXTIETH ANNUAL MEETING

Table 5

MORTALITY OF MEN RATED FOR OVERWEIGHT  
TOTAL CASES, BY AGE GROUPS AT ISSUE  
ACCORDING TO HEIGHT AND WEIGHT GROUPS

Ratio of Actual to Expected Deaths by Contemporaneous Mortality  
Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues  
of 1925 to 1934, Traced to Policy Anniversary in 1950

Age at Issue; Per cent departure from average weight	Per Cent Actual of Expected Deaths			
	All Heights	Short	Medium	Tall
All Ages, 20-64 years	150	151	145	166
Less than 30%	142	143	133	156
30-39%	151	148	148	177
40-49%	178	188	163	212
50-59%	234	190	280	226(12)
60-74%	282	266(10)	282(13)	*
Ages, 20-29 years	181	168	177	193
Less than 30%	219	—	—	219
30-39%	152	128(19)	147	176
40-49%	171	193(17)	143(24)	215(14)
50-59%	273	*	365(20)	*
60-74%	473(14)	427(5)	500(7)	*
Ages, 30-39 years	168	164	166	180
Less than 30%	145	130	129	170
30-39%	167	171	161	180
40-49%	218	179	241	228(19)
50-59%	251	200(14)	253	435(8)
60-74%	182(5)	*	*	0
Ages, 40-49 years	152	162	140	165
Less than 30%	148	151	141	162
30-39%	150	161	139	161
40-49%	172	216	125	234(16)
50-59%	215	201(18)	248(15)	*
60-74%	192(6)	*	*	0
Ages, 50-64 years	131	130	131	134
Less than 30%	131	138	127	124
30-39%	131	109	146	214(21)
40-49%	126	143(24)	114(22)	*
50-59%	133(5)	133(5)	—	—
60-74%	—	—	—	—

\* Less than 5 deaths.

Note: Figures in ( ) represent actual number of deaths in classes with 5 to 24 deaths. Classes with no experience most of which are outside Intermediate rating limits are indicated by —, and those with small experience and no deaths are indicated by 0.

## MORTALITY, INSURED OVERWEIGHTS 247

for the tall men in each weight group up to 50 per cent above average. Beyond that point, there is little experience on them, but it is somewhat worse than that for short men but better than that for men of medium height.

For all heights combined, the mortality ratios in the age groups up to 50 likewise show, with certain exceptions, a tendency to rise with increasing departure from average weight, but at ages 50 to 64 all the weight groups show pretty much the same excess mortality.

In the separate classifications by height, the upward trend with increasing weight still appears but the exceptions are more numerous. The experience has been particularly poor among the very obese men at ages 20 to 29.

If we compare the mortality ratios by height classes in the separate age and weight groups, we find that at ages under 40 the experience is least favorable for tall men. Above that age, the experience on them is on the average about the same as on those of short men. In general, at ages over 30 and for weight groups up to 50 per cent above average the experience is least unfavorable on men of medium height.

The experience on cases limited to Special Class has been worse than on those granted Intermediate insurance in most of the detailed groups by weight class, age and height. This may, in part, indicate that even within the weight groupings we have used, the average degree of overweight was higher in those cases, but other factors are probably involved.

Among women, too (Table 6), the aggregate experience at all ages and all heights combined shows a rise in the mortality ratios with increasing departure from average weight up to 60 per cent overweight, but the rise is very gradual. The experience on the most obese has been relatively favorable. Among the medium height and tall women, there is a fairly consistent upward progression of the ratios with increasing weight, but among the short women the ratios are virtually identical up to 50 per cent overweight, with a sharp rise in the next weight group followed by a drop to the lowest

## SIXTIETH ANNUAL MEETING

Table 6

MORTALITY OF WOMEN RATED FOR OVERWEIGHT  
TOTAL CASES, BY AGE GROUPS AT ISSUE  
ACCORDING TO HEIGHT AND WEIGHT GROUPSRatio of Actual to Expected Deaths by Contemporaneous Mortality  
Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues  
of 1925 to 1934, Traced to Policy Anniversary in 1950

Age at Issue; Per cent departure from average weight	Per Cent Actual of Expected Deaths			
	All Heights	Short	Medium	Tall
All Ages, 20-64 years	147	157	143	136
Less than 30%	139	155	132	127
30-39%	148	158	146	133
40-49%	156	158	155	157
50-59%	175	190	165	188(17)
60-74%	143	126(11)	162	*
Ages, 20-29 years	133	102	137	169
Less than 30%	0	0	-	-
30-39%	99	61(6)	104	132(9)
40-49%	144	138(13)	148	141(8)
50-59%	191	121(5)	196(18)	292(7)
60-74%	180(11)	*	216(7)	*
Ages, 30-39 years	152	162	151	130
Less than 30%	141	160	146	81(8)
30-39%	151	163	149	131
40-49%	150	130	158	169(18)
50-59%	197	253	171	177(6)
60-74%	146(19)	229(8)	126(11)	0
Ages, 40-49 years	150	160	145	145
Less than 30%	141	150	134	153
30-39%	159	168	156	141
40-49%	163	191	148	156(16)
50-59%	159	148(14)	181	*
60-74%	124(9)	*	206(7)	0
Ages, 50-64 years	138	157	129	118
Less than 30%	136	161	124	111
30-39%	138	149	133	120(14)
40-49%	169	160(17)	179	147(5)
50-59%	*	*	0	*
60-74%	0	0	0	0

\* Less than 5 deaths.

Note: Figures in () represent actual number of deaths in classes with 5 to 24 deaths. Classes with no experience most of which are outside Intermediate rating limits are indicated by -, and those with small experience and no deaths are indicated by 0.

figure in the small experience on those 60 to 74 per cent overweight. In all of the groups, except 60 to 74 per cent overweight, the mortality ratios for short women are the highest. The difference is most marked for the lesser degrees of overweight.

In the separate age groups, we find relatively sharp differentials in the mortality ratios with increase in weight among the medium height and tall women at ages 20 to 29, and to a lesser extent at ages 30 to 39. The experience on short women at these ages is rather inconsistent but has been unfavorable among the very obese at ages 30 to 39. At ages 40 to 49, the usual increase is found among the medium height women except at one point, and in the short women up to 50 per cent overweight, beyond which there is little experience. The tall women in this age group show little variation in mortality according to weight class. At ages 50 to 64, the experience is limited to weight groups under 50 per cent overweight. Only on the medium height women is it sufficient to show a trend, and among them the tendency for the mortality ratios to rise with increasing weight is apparent.

Comparing the mortality ratios according to height in the detailed groups, we find that short women past 30 tend to have the highest mortality, with some exceptions. There is no consistent difference between the medium height and tall women.

When the results according to rating class are considered, the ratios in comparable categories are for the most part higher for those limited to Special Class than for those granted Intermediate insurance. As in the case of men, this may reflect small differences in the degree of departure from average weight within the weight groups and minor factors in the medical history, as well as underwriting judgment.

#### *Causes of Death*

As we have indicated earlier, the mortality comparisons by causes of death represent careful estimates for attained ages 25 to 74 years, but these give, in our judgment, a fairly reliable measure of the major differences between these overweights

and standard risks. Unmistakably, the excess mortality of these overweights is largely accounted for by the high death rates from the degenerative diseases of the heart, arteries and kidneys, diabetes, and certain disorders of the liver, biliary tract and bowels.

Let us look first at the facts with regard to men (Table 7). The cardiovascular-renal diseases accounted in the aggregate for 50 per cent of all the deaths. The mortality from these conditions is approximately 1½ times that expected on the basis of standard experience. For chronic diseases of the heart, including coronary artery disease and angina pectoris, the observed mortality was about 40 per cent higher than among standard risks; for cerebral hemorrhage approximately 60 per cent; and for chronic nephritis approximately 90 per cent above the expected. The mortality ratios for these causes were significantly higher at the younger attained ages than at the older ones.

Diabetes showed relatively the greatest excess mortality among the major causes. The deaths from this condition were approximately 4 times the expected. Other conditions from which the mortality was double or more than that of the standard were cirrhosis of the liver, appendicitis and gallstones. Mortality from hernia and intestinal obstruction was 1½ times the expected. The mortality from cancer, all forms, and leukemia and Hodgkin's disease among these overweight men was not found to be excessive, although possibly our mortality standard for this cause is not accurate enough. However, when cancer mortality was analyzed according to site, the number of deaths from cancer of the liver and gall-bladder appeared to be significantly above the expected. The mortality from pneumonia was about the same as the expected. Diseases for which the mortality was low were tuberculosis and ulcers of the stomach and duodenum. The number of deaths from tuberculosis was little more than one fifth of the number expected by our standard.

Mortality from accidents was in the aggregate slightly higher than expected but the difference was not statistically

## MORTALITY, INSURED OVERWEIGHTS 251

Table 7

## PRINCIPAL CAUSES OF DEATH AMONG MEN RATED FOR OVERWEIGHT. ATTAINED AGES 25 to 74 YEARS

Ratio of Actual to Expected Deaths According to Estimates of Contemporaneous Mortality Experience on Standard Risks  
 Metropolitan Life Insurance Company, Ordinary Department, Issues of 1925 to 1934, Traced to Policy Anniversary in 1950

Cause of Death	Deaths	Per Cent Actual of Expected Deaths
Principal Cardiovascular-renal diseases	1,867	149
Organic heart disease, diseases of the		
Coronary arteries and Angina pectoris	1,377	142
Organic heart disease	748	*
Coronary disease and Angina pectoris	629	*
Cerebral hemorrhage	247	159
Chronic nephritis	243	191
Cancer, All forms	385	97
Stomach	62	85
Liver and gallbladder	33	168
Peritoneum, intestines and rectum	103	115
Pancreas	19	93
Respiratory organs	39	78†
Leukemia and Hodgkin's disease	26	100
Diabetes	205	383
Tuberculosis, All forms	24	21
Pneumonia, All forms	98	102
Cirrhosis of the liver	96	249
Appendicitis	76	223
Hernia and intestinal obstruction	39	154†
Biliary calculi and other gallbladder diseases	32	152†
Biliary calculi	19	206
Ulcer of stomach and duodenum	30	67
Suicide	63	78
Accidents, total	177	111
Auto	76	131
Falls	32	131

\* Satisfactory basis for comparison not available.

† Based on mortality rates on Standard risks for 1935-1939.

Note: Percentages in italics indicate statistically significant deviations from experience on Standard risks.

significant. However, for automobile accidents there was a significant excess. Mortality from suicide was below the expected.

The overweight women also show a large excess mortality from the degenerative diseases (Table 8). The number of deaths from these causes was about 1½ times the expected by

Table 8

PRINCIPAL CAUSES OF DEATH AMONG WOMEN RATED FOR OVERWEIGHT. ATTAINED AGES 25 to 74 YEARS

Ratio of Actual to Expected Deaths According to Estimates of Contemporaneous Mortality Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues of 1925 to 1934, Traced to Policy Anniversary in 1950

Cause of Death	Deaths	Per Cent Actual of Expected Deaths
Principal Cardiovascular-renal diseases	1,103	177
Organic heart disease, diseases of the		
Coronary arteries and Angina pectoris	697	175
Organic heart disease	515	*
Coronary disease and Angina pectoris	182	*
Cerebral hemorrhage	226	162
Chronic nephritis	180	212
Cancer, All forms	476	100
Stomach	34	86
Liver and gallbladder	46	211
Peritoneum, intestines and rectum	93	104
Pancreas	21	149
Breast	81	69
Genital organs	132	107
Uterus	103	121
Leukemia and Hodgkin's disease	23	110
Diabetes	235	372
Tuberculosis, All forms	20	35
Pneumonia, All forms	78	129
Cirrhosis of the liver	32	147
Appendicitis	41	195
Hernia and intestinal obstruction	31	141†
Biliary calculi and other gallbladder diseases	70	188†
Biliary calculi	50	284
Puerperal conditions	43	162
Suicide	23	73
Accidents, total	74	135
Auto	27	120

\* Satisfactory basis for comparison not available.

† Based on mortality rates on Standard risks for 1935-1939.

Note: Percentages in italics indicate statistically significant deviations from experience on Standard risks.

the standard used. As in the case of men, the greatest relative excess mortality was recorded for diabetes—about 3½ times the standard. The mortality from gallstones was nearly 3 times the expected, and it was high also for other biliary tract disorders, appendicitis, cirrhosis of the liver and hernia and intestinal obstruction.

Cancer mortality among these overweight women appears in the aggregate to be on about the same level as among standard risks. Mortality from leukemia and Hodgkin's disease is also about average. As in the case of men, however, there appears to be a significant excess for cancer of the liver and gallbladder. Mortality from breast cancer was low.

The death rate from puerperal conditions was greater than expected, and even though we cannot make comparison by the more accurate measure of deaths per thousand live births, our finding is in conformity with clinical experience. The mortality from pneumonia also was somewhat greater than the expected, but that from tuberculosis was low—only about one third the expected. Accident mortality was above the standard, even though the death rate from this cause is generally low among women.

#### IV. *Effect of Change of Weight*

A question that has often been put to us in the course of the Company's Weight Control Program is "What proof is there that weight reduction actually improves the outlook for the individual?" This is one of those questions for which it is rather difficult to obtain the kind of information that would provide an answer. Reasoning on this matter has been based upon inference from the facts on the general body of data regarding the influence of build and mortality and on clinical improvement following weight reduction by overweight persons with various disorders. It occurred to us in planning the present study that we could get some evidence on the matter from a study of those cases which subsequent to the issue of the substandard insurance received lower ratings, and in many cases standard insurance, because they

lost enough weight to qualify them for a lower premium class. We have considered separately those cases originally insured in our Intermediate class and those originally insured as Special Class, excluding Special Class B risks. Because of the difficulty of properly evaluating the mortality in the few cases where the change was made after age 64, we have excluded them. It should be noted in passing that in most cases the change was made as a result of the issue of new insurance in the lower premium class, with consequent revision of the rating on the earlier insurance.

Our findings are interesting (Table 9). Among the men originally limited to Intermediate insurance, the mortality subsequent to the change of rating, based on 129 deaths, was 113 per cent of the expected by the standard used. This may be compared with 142 per cent in the general experience on Intermediate cases in this study. Even better relatively was the result on cases originally limited to Special Class. Based upon 36 deaths, the mortality was 109 per cent of the expected. The facts by age relate to the age at which the weight reduction was officially recorded and on this basis we found that the greatest improvement was among the men past 40 in the group originally limited to Intermediate.

Our experience on women is rather limited largely because a smaller proportion of them own more than one insurance policy and consequently relatively few are rerated. Nevertheless, in the small experience on women originally limited to Intermediate who later qualified for standard insurance because of lower weight, the mortality ratio, based on 18 deaths, was only 90 per cent as compared with 142 per cent for the general experience on the overweight Intermediate cases. Similarly, the mortality ratio for those originally limited to Special Class, was 135 per cent, based on 15 deaths. There is no indication that the improvement in mortality was limited to the early years following the change.

#### *V. Discussion*

It is clear from the study that overweights as a group continue to experience a mortality exceeding that of persons of

## MORTALITY, INSURED OVERWEIGHTS 255

lighter weight, and this holds regardless of sex and age. The degree of excess mortality varies with a number of factors, but is significant throughout. Of prime importance is the

Table 9

### MORTALITY OF PERSONS RATED FOR OVERWEIGHT WHO SUBSEQUENTLY RECEIVED LOWER RATINGS AFTER REDUCTION IN WEIGHT, COMPARED WITH MORTALITY OF ALL CASES IN ORIGINAL RATING CLASS

Ratio of Actual to Expected Deaths by Contemporaneous Mortality  
Experience on Standard Risks  
Metropolitan Life Insurance Company, Ordinary Department, Issues  
of 1925 to 1934, Traced to Policy Anniversary in 1950

Age and Duration†	RERATED CASES					ENTIRE EXPERIENCE		
	Originally Intermediate			Originally Special Class		Per Cent Actual of Expected Deaths		
	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths	Years of Life Exposed	Deaths	Per Cent Actual of Expected Deaths	Intermediate	Special Class and Special Class B
<b>M A L E</b>								
All Ages, 20-64	14,681	129	113	4,236	36	109	142	179
20-29	1,682	6	168	465	1	*	163	231
30-39	6,100	39	144	1,697	6	82	152	220
40-49	4,985	57	124	1,503	16	115	146	172
50-64	1,914	27	72	571	13	121	128	143
1-5 Yrs.	6,025	36	114	1,847	10	96	125	155
6-15 Yrs.	7,687	75	110	2,171	19	97	144	178
16-25 Yrs.	969	18	126	218	7	249	150	196
<b>F E M A L E</b>								
All Ages, 20-64	3,619	18	90	1,827	15	135	142	161
20-29	447	—	—	138	1	*	127	154
30-39	1,569	4	*	808	6	206	144	174
40-49	1,161	5	68	588	5	127	146	162
50-64	442	9	142	293	3	*	136	147
1-5 Yrs.	1,623	8	118	810	6	167	141	175
6-15 Yrs.	1,855	9	76	927	7	109	141	151
16-25 Yrs.	141	1	*	90	2	*	142	169

\* Less than 5 deaths.

† Computed from date of rerating. Entire experience on basis of date of issue.

indication from this study that even for moderate overweights the mortality relative to standard risks has increased in recent years. This is confirmed by the fact that for the overweights in our study who were limited to Intermediate insurance, and who may be characterized as essentially moderate overweights, the mortality ratio has been running appreciably higher than the general experience in that branch of the business. Evidence along the same lines is found in the recent studies of the Provident Mutual and London Life on build and mortality. The former, particularly in its comparison of the experience in the period 1937-1947 with that for 1920-1937, showed definitely a relative increase in mortality of moderate overweights as against a relative decline in the mortality of underweights.

We are inclined to attach particular significance to the fact that the mortality ratios for men at the younger ages are in the aggregate as high or higher than at the older ages, except in the early years after issue. Among women the situation differs somewhat in this regard, but even so, mortality is appreciably above normal among young overweight women. It is apparent then that overweight *per se* no longer can be considered advantageous, even for young adults. While it is true that tuberculosis mortality remains lower in these overweights than in persons of lighter weight, this is a minor consideration in the whole problem because of the drastic decline in recent years, both absolutely and relatively in the importance of tuberculosis as a cause of death, and also because, among men, the peak of mortality from it has shifted steadily to the middle and older ages.

In like manner the mortality from pneumonia has declined very sharply, particularly within the last decade, and prevention and treatment of this acute disease affords far better protection from it than extra poundage, even for young people. Beyond that, when pneumonia strikes, the extra pounds are of no particular advantage. It is noteworthy that the mortality from pneumonia among the overweights is fully as high as in the standard experience.

The major problem today in insurance medicine and in public health is the increasing predominance of the degenerative diseases as causes of disability and death, and the findings of this study emphasize anew the association of overweight with these diseases. These new statistics provide up-to-date evidence on the matter which should satisfy any doubting Thomases. Obviously the study does not give reliable clues as to the nature of this association; but whether it is primarily a mechanical effect of the extra work put on the heart and circulation or whether it represents a fundamental metabolic defect, or both, the fact remains that the association exists.

On one special aspect of the matter, the occurrence of coronary attacks in younger people, recent clinical papers raise the question as to whether the proportion of overweights is unduly large (11, 12). The data they offer appear to show no such excess. Time does not permit us to go into detail regarding this matter, but from our examination of the published data we are somewhat skeptical as to how representative the material is. Evidence has also been presented that the young coronary victim tends to be stocky rather than overweight (13). We have looked for evidence regarding this in overweights in the present study. We have tabulated the heights at examination of the men dying from coronary disease and angina pectoris. We found the height distribution to be essentially normal in this group of cases.

Our analysis shows too that the overweight is subject to a wide variety of digestive and metabolic disorders. It is interesting to note the several disorders of the liver and biliary tract from which the mortality is high—cirrhosis of the liver, gallstones, and even cancer of the liver and gallbladder. In addition, the mortality among overweights remains notoriously high for diabetes. There is also an excess mortality from hernia and intestinal obstruction.

While the results of life insurance studies are unequivocal with regard to mortality of overweights, the build classification on which they are based has been questioned because it is dependent largely on average weights for height, and

we know this is a weak reed to the extent that it fails to reflect the normal variations in skeletal and bodily proportions and in muscular development. Parenthetically, we may say that the rating standards in this study are so generous that virtually all the cases included by us would be overweight in greater or less degree by any definition.

Various methods have been proposed for estimating proper weight in relation to body structure (14, 15, 16, 17) but have not won wide acceptance, nor are they practical for use either by laymen or for research projects involving large numbers of people. They are not applicable in insurance work because the detailed measurements they require are not available in insurance records, or the procedure is unsuitable for other reasons. Consequently, insurance studies of build and mortality have relied on gross figures of height, weight, and girth. The most notable departure from this was the set of studies based upon the ratio of spine length to height, an adaptation of Dreyer's concept, which was used in a study of overweights by the Association 25 years ago (18, 19, 20). The investigation was not very productive but, even so, was made possible only because the Union Central Life Insurance Company had included sitting height as one of the measurements in its record of applicants at one time. Mention should be made also of Clark's papers on the use of Davenport's Index of Build ( $\frac{w}{h^2}$ ) (21, 22), but his work did not involve classification and tabulation of original data on the basis of this index. Until, therefore, new and simpler methods can be developed for estimating optimal weight, and more accurate and detailed measurements are made of insurance applicants, we are not justified in modifying our insurance practice by introducing estimates of build characteristics. This may have value in popular health education. We have developed our so-called Ideal or Desirable Weight Tables (23, 24) for the latter purpose and these are now in extensive use. Nevertheless, we in the insurance business should attempt to develop better weight standards than we now have, in cooperation with outside investigators who are interested in the problem, with a view to more accurate ratings for build.

Perhaps the most encouraging feature of this new study is the evidence it affords that weight reduction pays, as shown by the lower mortality experienced among those substandard cases in our study who subsequently received a lower rating following loss of weight. It is true, of course, that we do not have the entire weight history of these individuals and it is not unlikely that many of them subsequently regained some or all of the weight lost. As against this, it should be noted that contrary to the tendency of weight to increase with age, these people had definitely taken off weight and, in some instances, the change was recorded many years after the issue of the policy which was included in the study.

The findings of this study have distinct application for public health as well as for insurance and clinical medicine in demonstrating that overweights, regardless of the benefits of modern medical and public health advances, still experience higher mortalities than persons of lighter build and that this is due to the higher frequency or earlier development of degenerative diseases, diabetes, and certain other conditions. The study puts on a firm basis our educational program for weight control. It confirms the numerous clinical evidences of the disadvantage of overweight, as well as the benefits of reduction of overweight. We are all the more confident then that in promoting efforts along these lines in our Weight Control Program, which will feature our health work on a national scale this coming year, we are rendering the nation as well as the insurance business a real service. We hope that the members of the Association will give this work active support. It has already received the endorsement of the American Medical Association, The American Heart Association, The American Diabetes Association, and other national and medical health agencies, both voluntary and official.

#### VI. *Summary and Conclusions*

A new mortality study of overweights insured in the Metropolitan Life Insurance Company, based on cases limited to substandard insurance solely because of overweight during the years 1925 to 1934 and traced to 1950 shows that these overweights, virtually all of whom were at least 20 per cent

above the average for their height and age, have a mortality significantly above that of standard risks and that in varying degree there is an excess mortality regardless of sex, age, or height. Among the men, the mortality ratios tended to be greater at the younger ages at issue than at the older ages, and to rise with increasing duration of insurance. Among women, however, the variations in both these respects are relatively small.

The study also shows that the mortality tends to rise with degree of overweight. This is particularly marked among the younger overweights. The study provides up-to-date facts on the variety of diseases, vascular, metabolic, hepatic, and other, which are responsible for the excess mortality of overweights.

A feature of the study is the experience on those overweights who brought down their weight and were subsequently granted insurance at lower rates of premium. This is perhaps the best evidence produced to date that there is long-range benefit from weight reduction and this should support our public health propaganda for weight control. Certainly as matters stand today, this appears to be the only practical approach to the problem of preventing or retarding the degenerative diseases of middle and later life that now far outrank all other diseases as a cause of death.

Our detailed results will be useful in reappraising our practices with regard to rating of overweights. To some degree, the results that we have presented understate the relative mortality of overweights today. The decline in mortality from pneumonia and other infections which chiefly affect underweights has accelerated since 1937, and consequently the full effect of this on the mortality of overweights as compared with those of lighter build is diluted in an experience which covers the entire period since 1925.

We are aware of the many inconsistencies in our detailed findings. Part of our difficulty stems from the relatively small amount of material at our disposal in the more obese groups. Part arises out of the fact that we have confined

ourselves to cases limited to substandard on the basis of our rating standards which were not uniform with respect to percentage departures for average weight. We readily admit, therefore, that we have not produced the definitive study on overweights. In fact, it may not be possible to do this on the basis of insurance experience alone because our records contain only a few crude physical measurements, and these frequently are inaccurate. The best way out of this difficulty is to combine our resources with those of clinical investigators and anthropometrists in setting up some good follow-up studies on persons for whom better criteria of overweight are used.

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## MORTALITY, INSURED OVERWEIGHTS 263

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PRESIDENT YLVISAKER — Thank you very much, Dr. Dublin, for the studies you have directed, for presenting your results here, and for emphasizing the significance of overweight so well to us. I would also like to thank Dr. Dublin for all the statistical studies that he and his office publish, which are of value to all of us. I personally read the Metropolitan Statistical Bulletin from beginning to end every month, and I think there is no statistical health publication which is more frequently quoted. This mortality study was brought to my attention by Dr. Bonnett, Medical Director of the Metropolitan Life Insurance Company and I have asked him to comment further on the underwriting significance of this study.

DR. BONNETT — Dr. Ylvisaker, Gentlemen: From my observation of the audience reaction, Dr. Dublin's paper has been received with the same keen interest with which I read it.

Overweight is perhaps the commonest impairment which confronts the underwriter and the one which most frequently leads to an appeal when a rated policy is issued. The appeals, as you know, are standard—this man is unusually big-boned, all the weight is muscle, he is active, he has never had a sick day in his life; his father, grandfather and great-grandfather were all big-boned and heavy, and all lived to 75 or more. Frequently, in such cases, we are hard put to it to justify the fact that one pound more or less for the height means a difference between standard and substandard insurance, or various classifications of substandard.

There is great need for a study which will be a measure of the "pure" excess mortality because of overweight. Unfortunately, Dr. Dublin's study does not give us this pure factor, and the reason is that the cases studied were not taken from a single base line. During the years covered by Dr. Dublin's study, it was the custom in the Metropolitan to have individuals age 45 or less applying for not more than \$2,000 examined on a short form. This examination did not include the blood pressure or urinalysis unless there was clear indication for it at the time of the examination or it was subsequently requested by the Home Office. Approximately two thirds of our substandard policies in those years were issued on that basis. Moreover, the weight was ordinarily estimated by the examiner and, except when the Clark table came into use as a check on estimated weights by reported measurements, many of these individuals undoubtedly received lower ratings than their actual weight would have called for. As I understand it, these are included in this study, as well as the individuals whose weight was carefully determined by tape and scale at the time of the examination, or whose physical examination included a blood pressure and urinalysis. It is likely that this is responsible for part of the excess mortality in the younger groups, 20 to 29 and 30 to 39, in the men who in those years made up the majority of applicants for insurance in the Metropolitan. Another reason for the high relative mortality at the younger ages is that the mortality of young overweights apparently has not improved as rapidly as general mortality at these ages.

## MORTALITY, INSURED OVERWEIGHTS 265

As Dr. Dublin pointed out, the most dramatic item is evidence that a reduction in weight, if maintained for a significant period, does materially decrease the excess mortality. Doctors have been looking for clear evidence of this for a long time and I have no doubt that this experience, although relatively small and admittedly not complete, will be welcomed by practicing physicians as proof that their advice to reduce is well based. We do not know how or why the weight of these individuals was reduced, nor how significant or sustained is the value of voluntary reduction. This aspect of the problem might well be studied by other companies, and perhaps more in detail, and possibly among employees, to give physicians and public health authorities added arguments in favor of weight reduction.

The causes of death among both the men and women are not at all surprising when we consider the method of selection and the presumed effect of overweight, if not in the production at least in the enhancement of these diseases. Some of the persons with hypertension, nephritis, and diabetes may have been missed because of our failure to analyze urine and take blood pressure at the time of issuing the insurance. This may also be part of the explanation of the high rate of liver disease.

I am more at a loss, however, to understand the high rate of deaths from gallstones, biliary tract disorders, appendicitis, hernia and intestinal obstruction. We know the tendency of overweight people to develop gallbladder disease, but there is no reason I can see for a high death rate among them unless it be the old assumption that overweights were simply poor operative or surgical risks. This may or may not be the case today, but it was a widely held belief in my early days as a surgeon.

Perhaps that has all changed now and there may be proof somewhere that a fat person without organic disease is a happy, healthy individual. I should like very much, however, to see a concerted effort to prove or disprove this thesis, and with that end in view I should like to recommend to the Medical Directors' Association and also to the Society of

Actuaries a study, in the not too distant future, of the effects on mortality of pure overweight. This means that the cases studied will have to be selected from a common base line—physical examination, blood pressure and urinalysis. If three groups were studied, we might get a measurement of the mortality due to overweight alone. These groups would be:

- (a) Individuals granted Ordinary insurance, standard or substandard, on a simple physical examination without blood pressure or urinalysis in which the weight is estimated by the examiner or is the figure given by the applicant or agent.
- (b) Individuals whose medical examination includes blood pressure and urinalysis, and also the exact height and weight at the time of the original examination.
- (c) A group with the same basic medical examination as in (b) but in which the height and weight are estimated by the examiner or based on the applicant's statement.

I believe that such a study is now feasible among the companies which are contributing to the large mortality investigation currently under way under the auspices of the Society of Actuaries. Since the study would be a measure of the excess mortality because of overweight alone and, therefore, a guide for the underwriting of such risks in the future, it should be based on fairly recent experience, probably over the past ten years.

PRESIDENT YLVISAKER—Thank you, Dr. Bonnett. I am afraid we shall have to refer your recommendations back to you as chairman of the Mortality Committee.

Our next speaker is Professor of Medicine at Temple University Medical School in Philadelphia. He is a wonderful teacher and clinician and one of the leaders in Philadelphia medicine. Among other activities, he is at present also Director of the Post Graduate Institute of the Philadelphia County Medical Society. He is very much interested in our overweight problems. I am delighted to introduce Dr. Thomas Durant to you. He will discuss dietary factors in the development of atherosclerosis.

## INSURANCE HAZARDS OF OVERWEIGHT DIETARY FACTORS IN THE DEVELOPMENT OF ATHEROSCLEROSIS

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*Temple University Medical School  
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It is a real pleasure for me to be with you here today to discuss a subject in which I have been tremendously interested for some years. From a purely quantitative standpoint, atherosclerosis is one of the major, if not *the* major, public health problem in the United States today. Hence, attempts to solve the intricacies of its etiology are of paramount importance. Furthermore, like the game we used to play when we were children in which the music sounded louder when we got close to something we were hunting, I think that in research today the music is sounding much louder in relation to an understanding of the etiology of atherosclerotic disease.

I would like, first of all, to be certain that we know whereof we are speaking today when we refer to atherosclerosis. It is because this morbid process is a disease of the intima that it is so serious. It impairs the nutrition of the organ supplied, and the coronary arteries have a special predilection for the disease, the reason for which may be a part of the special mechanical factors involved in the coronary circulation. The impairment of blood supply to the myocardium becomes a serious factor in determining whether or not a person will survive.

The age factors in this disease are of considerable interest because atherosclerosis is not a disease of the elderly alone. It involves persons at the height of their usefulness as citizens. It seems to be increasingly frequent among the young age groups. Certainly the army experience has been of tremendous interest in this respect in showing the amazing amount of coronary disease among very young individuals.

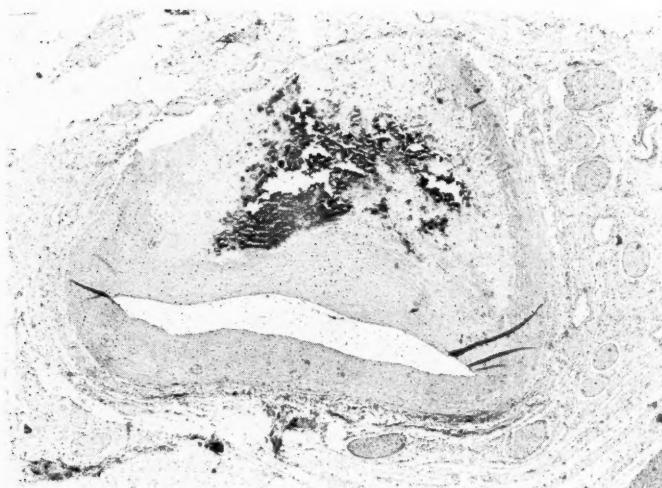


Figure 1

Section of anterior descending branch of left coronary artery of male, aged 26. Note large atheroma with numerous cholesterol clefts and an area of calcification.

The sex incidence is of great interest. In the younger age groups, there is a marked predominance of involvement of the male sex, the ratio in the twenties being 24 to 1, male to female. By the time we get into the fifties, the ratio is about 3½ to 1. When we reach the seventies, the sexes are about equal in incidence. There are undoubtedly some very important secrets hidden in this sex difference which, when they are understood, will be of tremendous practical importance. At the moment, as Dr. Page has put it, it is simply another example of the confounded superiority of the human female.

There are some very interesting constitutional factors in relation to atherosclerotic disease. The typical mesomorphic individual is unusually susceptible to coronary artery disease, while the endomorphic individual is intermediate in his tendency toward it. The ectomorphic person, on the other hand, is blessed with a certain amount of apparent immunity to the disease. This constitutional relationship has been

shown pretty definitely to have statistical significance. It does not mean, of course, that an ectomorphic person cannot develop coronary artery disease since there are mixtures of constitutional types, but it does mean that the predominant tendency is in the direction of immunity.

It is of further interest that Dr. Gertler and his group (1) have shown recently that the mesomorphic group is the group most likely to have disturbances of lipid metabolism.

Psychosomatic factors have been brought into this picture as well as into that of many other disease states. There is no question about the fact that emotional crises play a very important part in the *complications* of the disease. We are all aware of the fact that a person with angina pectoris quickly learns the painful consequences of a fit of anger or of any other epinephrine-producing emotional disturbance. However, the question as to whether or not the disease is *primarily* related to emotional factors is another story.

There is a psychologic type that does tend to develop coronary artery disease. To quote Dr. Flanders Dunbar (2), these are predominantly extroensive individuals. They have a distinguished appearance and considerable evidence of control and surface calm, with little evidence of strain. They have an air of self-sufficiency with a tendency to dominate in social relationships through superior argumentative skill. Their educational record is unique in that there is a tendency to complete whatever unit of work is undertaken. Also, there is a tendency toward large families and a high marriage rate. "Accepting the idea of hierarchy, these persons identify themselves with authority figures and strive to become superauthorities."

But to say that the average coronary patient is this type of individual, psychologically speaking, does not mean that this is, necessarily, a primary etiologic factor. We might argue that this type of individual is likely to be successful in life and that, therefore, he does better at the dinner table than the citizen who does not have so much success.

That brings us to a consideration of the metabolic factors in relationship to atherosclerosis and coronary disease. The interest in this relationship goes back to the demonstration by Anitschkow early in this century that atherosclerotic disease could be produced in the rabbit by the feeding of cholesterol.

Since that original work, there has been a tremendous amount of vital information collected on this subject (3). And, as the years have gone on, there has been a decreasing number of persons who refer laughingly to "the cholesterol disease of the rabbit", inferring thereby that what happens in the herbivorous rabbit cannot be carried over to omnivorous man and that it has little relation to our human problems. Those who hold to a metabolic defect in atherosclerotic disease do not deny that there may be, and probably are, very important local factors in the artery which enter into this story. But they feel that the local factors are definitely enhanced by the abnormal metabolism.

Time does not permit a review of the evidence supporting the existence of metabolic relationships in this disease. This evidence, however, can be summarized under several headings. First, the geographic incidence of the disease points toward an apparent relationship between fat intake and atherosclerosis. Second, the effect of war with its marked demands for fat (the god Mars demands fat as much as he does blood) has shown that those nations which have been involved in prolonged wars and which have, therefore, had to divert fat stores from the dinner table to maintenance of the war machine, have had a striking diminution of coronary artery accidents during that period, even though the psychologic phenomena of such periods are much greater than they are during peacetime.

Third, there is the fact that atherosclerotic disease has a very definite relationship to all of the diseases known to be associated with impaired lipid metabolism. These include such conditions as diabetes, myxedema, nephritis, and xanthomatosis. All of these are associated with premature atherosclerotic tendencies. Perhaps the one exception is biliary

cirrhosis in which the blood cholesterol is markedly elevated and in which, according to some studies, there may not be the same tendency to coronary disease. Dr. Ahrens (4) has attempted to explain this on the basis of a tendency in this disease to maintain a fairly normal phospholipid-cholesterol ratio despite a marked absolute rise in the level of cholesterol.

Fourth, there is the animal experimental work which has shown that all groups of animals, whether they are herbivorous, carnivorous, or omnivorous, can be made to develop atherosclerotic disease if the appropriate disturbance of lipid metabolism can be induced in the particular animal. It is much easier in the herbivorous group, but it is very easy in certain omnivorous animals such as the chick in which very premature atherosclerosis can be produced by the feeding of a high cholesterol diet. The carnivorous group are the most resistant, but even in them lesions practically identical, if not totally identical, with those in the human coronary artery can be produced by giving thiouracil and cholesterol.

In human studies it has been found that, on the average, there is a tendency toward elevated cholesterol among persons with coronary artery disease, but there are many exceptions. That there are these exceptions has been a bit disturbing over the years to those who hold to the metabolic theory. A further disturbing fact has been the failure of a very low cholesterol diet to influence the blood cholesterol level or the atherosclerotic disease process.

A great deal of recent study of cholesterol metabolism has attempted to answer these difficult questions. The use of "tagged" cholesterol and cholesterol precursors has been possible recently and this has added much to an understanding of lipid metabolism. The absorption of cholesterol is definitely influenced both by fatty acids and pancreatic esterase. Fatty acids, of course, may also be absorbed in the free form.

In the liver, very important things happen. Some of the cholesterol is changed to cholic acid, and that is secreted again into the gut via the biliary system. Some appears in the blood stream as free cholesterol and some is esterified in the liver

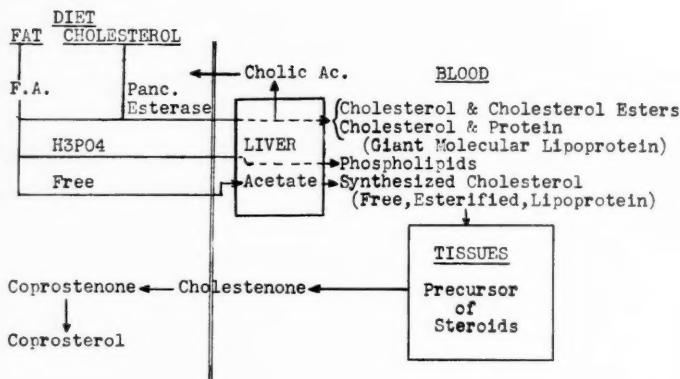


Figure 2

Schematic representation of cholesterol metabolism. The vertical double line represents the intestinal wall.

before appearing in the serum. The formation of lipoprotein complexes is of great importance, and we are going to emphasize this since it appears that the ability of the individual to form the right kind of lipoproteins in the blood stream is one of the very important factors in the avoidance of this disease.

The phospholipids may very well have an important relationship to this whole story. Unfortunately, at the present time we are unable to influence in any way the phospholipid-cholesterol ratio by the feeding of lipotropic substances.

One of the reasons that it has been impossible to influence the blood cholesterol level by simply cutting down on the dietary intake of cholesterol is the fact that, as long as there are available the basic acetate building blocks from the fat of the diet, the body can and does synthesize cholesterol. It does a beautiful job of it, even though we keep the intake of cholesterol down to a very marked minimum. It is, then, the *endogenous* cholesterol metabolism which has defeated all attempts to reduce blood cholesterol levels by dietary restriction of this particular lipid.

To follow through with the diagram, the various forms of cholesterol are carried to the tissues and there the lipid becomes the precursor of steroids. This is one of its very important functions in normal metabolism. Eventually, cholesterol is broken down partly in the tissues, partly in the gut, to cholestenone and then coprosterol. There are some who feel that there may be a disturbance in the breakdown of cholesterol which influences the cholesterol blood level. That is a subject for future investigation.

I would like to emphasize two things in particular concerning dietary fat and cholesterol metabolism. First of all, there is the important relationship of ingested fatty acids to the absorption of cholesterol from the gut. Second, the dietary source of fatty acids provides acetate building blocks with which the liver can synthesize cholesterol even though there is a low dietary content of the latter. Thus it can be seen why a very *low fat*, low cholesterol diet is effective in producing a reduction of the blood cholesterol level when a low cholesterol diet alone fails to do so. The latter type of diet cannot be made more pleasant for the patient by supplying him with vegetable fats to take the place of the animal fats that have been denied him.

It would have been much easier for the proponents of the lipid theory of atherosclerotic genesis if it had been possible for them to claim an elevation of the blood cholesterol level in all cases of atherosclerotic disease. This was not possible, however, since some cases, even among those with advanced lesions, have blood levels of cholesterol which are well within the normal range. Some have attempted to circumvent this difficulty by claiming that the accepted upper normal limit of blood cholesterol (250 mg./100 cc.) is too high and that the "safe" level is actually a much lower one. Others have pointed to the fact that there is considerable fluctuation of the level in persons with atherosclerosis which is not found in normals, and that single determinations, or even several of them, may not give a true picture of the lipid metabolic defect in any given case. Such arguments, however, have not provided a satisfactory explanation, and it has only been

since the discovery of the importance of physicochemical factors, not necessarily reflected in quantitative changes in the analytical blood levels, that the picture has become clarified.

In order to understand this phase of the metabolic picture, and the relationship of physicochemical factors to the deposition of substances in the arterial intima, it is important to refer to the studies of Hueper (5). This investigator found that the introduction into the blood stream of animals of inert macromolecular substances (e.g. polyvinyl alcohol, methyl cellulose, pure citrus pectin, gum acacia, etc.) results in the appearance of foam cells in the intima and lesions that resemble human atherosclerosis in every respect except that no fat is present. This type of intimal lesion he designated Macromolecular Atherosclerosis. Thus it was demonstrated that large molecules may not traverse the intimal elastic barriers to be carried away in the adventitial circulation, and that, furthermore, these deposited substances result in a reaction within the intima which resembles very closely (except for the character of the initiating substance) the reaction seen in human atherosclerotic disease.

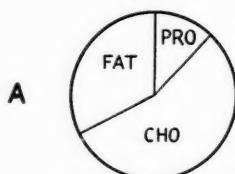
The stage was thus set for the epoch-making discoveries of Gofman and his associates (6). You will recall from the paper presented by Dr. Gofman last year that the ultracentrifuge has made possible a classification of lipid molecules circulating in serum according to their flotation rates, using Svedberg units ( $S_f$ ) as the means of designating these rates. In the serum of normal persons, lipids belonging to the class  $S_f$  3-8 are found. These are apparently innocuous. At the other extreme of  $S_f$  values are particles with values greater than 75. This class is greatly increased, even in the normal, following fat meals, and includes the macromolecules known as chylomicrons (7). These particles probably are not related to atherosclerotic disease, although this is not entirely proven as yet. In between these two extremes there are the  $S_f$  30-70 and  $S_f$  12-20 groups. The former constitutes the major fraction of alimentary lipemia and may or may not be related to atherosclerosis. The latter has been shown to be definitely

related to this disease, both in the human and in experimentally induced disease in animals.\* The molecules in the latter group have molecular weights of approximately 3,000,000, and contain about 30 per cent cholesterol and 5-10 per cent protein. The  $S_f$  3-8 particles of normal serum are higher in protein content, and this may be a very important factor in normality. In other words, the normal metabolic machinery may have the ability to use protein in the formation of lipoprotein complexes with low  $S_f$  flotation rates.

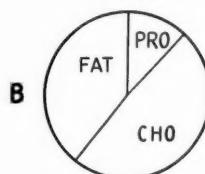
Happily, from a therapeutic standpoint, Dr. Gofman and his co-workers have shown (6) that the molecules related to atherosclerotic disease are considerably influenced, in the long run, by a dietary which is very low in fat (25-50 Gm. total of animal *and* vegetable fat per day). The majority of persons having molecules in the  $S_f$  12-20 class will show a decrease in the blood level of these molecules within one to eight weeks on such a diet. Such a dietary is not an easy one for the average American to follow; but, until such a day arrives that it is possible to influence the lipid metabolic defect of atherosclerosis more easily, and by non-dietary means, this type of diet will have to constitute one of our most important weapons against this disease. Heparin may possibly be the non-dietary answer to this problem (8), but it is too early to be sure of this at the present time.

This leads us to a discussion of the American dietary and its possible relationship to atherosclerosis. Does the average American eat a high fat diet, and has there been an increase in the consumption of fat in recent years to parallel the increasing incidence of coronary disease? In Figure 3, I have analyzed, on the basis of Department of Agriculture statistics, the per capita consumption of food in the United States, and have calculated the percentage of calories derived from the three basic types of foods. This has been done for the years

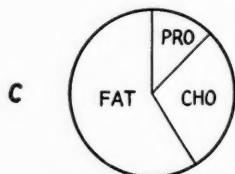
\*Dr. Ancel Keys (J. A. M. A. 147:1514, 1951) has indicated that some of the conclusions of Gofman and co-workers are to be criticized on the basis of statistical analysis. He points out that, "though both giant molecules and total cholesterol show distinct tendencies to be maintained in higher concentrations in the serum of persons with coronary disease than in clinically healthy persons, neither measurement is a good discriminator between such patients and healthy persons, and that, if there is any advantage to one measurement over the other in detecting or predicting coronary disease, the evidence is in favor of total cholesterol".

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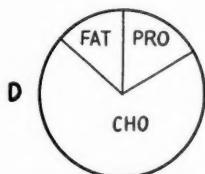
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**BUSINESS AND PROF. MAN**

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**THERAPEUTIC**

Figure 3

A and B: Estimated average American dietary for the years 1910 and 1948, respectively. The circles are divided in such manner as to represent the percentage of total calories supplied by each of the basic food groups. The figures below the circles represent the number of grams of each food group and total calories. (From Misc. Public, 691, U. S. Dept. of Agricult., Aug. 1949: Estimated nutritive value of civilian food supply, expressed as daily quantities of nutrients per capita, after allowance for waste and deterioration.) C: Calculated average daily dietary intake of a group of business and professional men. D: Diet prescribed for average patient with coronary disease who is engaged in a sedentary occupation.\*

1910 and 1948. The intervening years are not presented in the figure, but are intermediate in their data, with a definite trend from the 1910 figures to those of 1948. It will be noted that the protein and caloric intake for the two years has not changed appreciably, but that there has been a very real shift in the percentage of calories derived from fat as compared to carbohydrate. It is understandable that such a shift should occur, in a land of increasing prosperity, since good cooks, whose resources are in no wise limited, tend to rely heavily on fat to provide smoothness, delectability and satiety for the meals they prepare. Furthermore, when we consider the

\*Courtesy of the Editor, Annals of Internal Medicine.

dietary of the business and professional man (noted as they are for their tendencies toward coronary disease) it will be seen that their metabolic machinery is asked to handle an even higher intake of fat than is that of the *average* American, even though the total calories ingested are less in number (9). The dietary of such individuals, when compared to the therapeutic regime now in use in the treatment of coronary disease, provides a very striking contrast.

Thus we may say that the average American, especially the business and professional man, does eat a high fat diet, and that in recent years there has been an increasing trend toward fat utilization in the average dietary of our country. That such fatty dietaries do not influence all people alike is simply a manifestation of the fact that some are fortunate enough to inherit a metabolic machine which can handle such fuel without the production of "clinkers", whereas others are not so fortunate in their genetic endowment. Thus persons from non-coronary families are able to ingest large amounts of eggs, butter, cream, etc., throughout life and live to ripe old age without evidence of significant atherosclerotic disease. On the other hand, those with an unfavorable inheritance may, even on a moderate fat intake, develop coronary disease early in life. The family history is indeed a valuable guide in estimating the group to which any particular patient may belong.

Concerning the relationship of obesity to atherosclerotic disease, there has long existed a clinical impression that coronary disease is more prevalent in the obese than in the lean. It is surprising therefore, that Garn, Gertler, Levine and White (10), in a recent study of ninety-seven men who had experienced myocardial infarction prior to the age of forty, found that these men were not overweight when compared with a group of healthy men of comparable mean age, occupation and mode of living. Possibly the group selected is not a truly representative one, but the findings do emphasize the fact that non-obese individuals may be victims of atherosclerotic disease. Perhaps the inheritance of a genetic defect in metabolic machinery, sufficiently severe to result

in serious coronary involvement before the age of forty, may not require a very large fat and caloric intake to produce disease in the arteries and result in obesity as well. My own experience with coronary disease in the young is in accord with the findings of Garn, et al, it being not at all unusual to see young coronary patients who are of normal weight, or even somewhat underweight according to usual standards, having marked disturbances of cholesterol metabolism.

After the age of forty, obesity is a much more common accompaniment of coronary disease than it is in the young. Here, the effects of a high caloric intake with declining physical activity, or an actual increase in appetite associated with a decline in potentialities for non-caloric emotional satisfaction, tend to lead to obesity, and the oft associated high intake of fat leads to coronary involvement (if the metabolic machinery is impaired). The frequent association of hypertension and diabetes with obesity may also be an important factor favoring the association in this older age group. When coronary disease and obesity are present together, the increased load of the obesity adds to the difficulties under which the heart is laboring and unfavorably influences the course of the disease.

Thus, in conclusion, we may say that the evidence today points strongly to a lipid metabolic defect as an etiologic factor of prime importance in atherosclerotic disease, and that the American public, with its trend toward an increasingly rich dietary, is "playing into the hands" of this serious enemy of health and happiness. Until better means of prevention and treatment become available to us, the importance of limitation of fat intake is paramount, especially in those who are potential victims, as indicated by a coronary type of hereditary background.

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## INSURANCE HAZARDS, OVERWEIGHT 279

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PRESIDENT YLVISAKER — Thank you very much, Dr. Durant for this very instructive discussion.

The complications of overweight which we meet in our daily work are primarily diabetes and hypertension and it should be helpful to have discussions on these conditions and their relation to overweight. Dr. Robert L. Weaver and his associates in the Penn Mutual Life Insurance Company have been very cooperative and helpful, and two of their Assistant Medical Directors, Dr. Dillon and Dr. Trapnell, have agreed to prepare a discussion on "Overweight as a Contributing Factor in the Development of Diabetes and its Complications". Their paper will be presented by Dr. John M. Trapnell, another relatively new member of our Association. He is also appearing for the first time on our program.

## OVERWEIGHT AS A CONTRIBUTING FACTOR IN THE DEVELOPMENT OF DIABETES AND ITS COMPLICATIONS

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The association of obesity with diabetes has been recognized almost as long as the disease itself. In India during the fifth or sixth century the symptom complex of diabetes was described as occurring principally in the rich because of their overindulgence in carbohydrates such as rice, flour and sugar. An astute French physician noted the disappearance of glycosuria in some diabetics when food was scarce during the Siege of Paris in the Franco-Prussian War, and subsequently applied that observation to the management of his patients.

Allen (1), working with partially depancreatized animals in which enough gland was left to prevent their becoming diabetic, found that when they were made fat by overfeeding diabetes resulted. It was the result of his experimental work which put the dietary treatment of diabetes on a rational basis during the pre-insulin era. From the clinical standpoint, it was noted that the effects of dietary restriction were particularly good in the obese diabetic patient.

Newburgh (2) studied the effect of weight reduction upon the hyperglycemic curves of 62 obese men and women having an average age of 52 years. As a whole, these patients were relatively mild diabetics, who, however, had been referred for a number of reasons, including in some cases the statement by their physicians that diet and insulin had not abolished their glycosuria. Of the 47 patients who cooperated fully in undergoing adequate weight reduction, the glucose tolerance curves became normal in 77 per cent of the cases.

Joslin (3) analyzed data from one thousand successive cases of diabetes for whom the age, date of onset of the disease, and weight and height were known. Of this series 77 per cent were overweight, 15 per cent were in the standard weight zone and 8 per cent were underweight at or preceding the onset of their disease.

Variation From Normal of Maximum Weights at or Prior to Onset of 1,000 Cases of True Diabetes, Calculated for Height, Age and Sex.

Age (years)	Number of Cases	Percentage in normal average zone (+5 to -5 per cent)	Percentage of each decade Below standard weight	Percentage of each decade Above standard weight
0 to 10	43	37	44	19
11 to 20	84	39	29	32
21 to 30	112	19	10	71
31 to 40	172	6	5	89
41 to 50	244	12	3	85
51 to 60	252	12	1	87
61 to 70	79	10	6	84
71 to 80	14	14	7	79

Looking at the percentage above standard weight column, it is readily apparent that overweight is not a predominant finding in diabetic children. However, beginning with the early twenties and continuing throughout the span of life we see that obesity is present in the majority of those who develop the disease. Obviously, in the early decades growth is mainly in a vertical plane, whereas later it is horizontal. In the older individual the percentage overweight may be even higher than is indicated.

Brozek and Keys (4) have pointed out the limitations of so-called normal body weight as a criterion of normality. In older men, the amount of body fat (determined by the thickness of skin folds and specific gravity) is strikingly greater than in younger men. Actuarial concepts of normal body weight tend to obscure the marked changes of aging which involve not only an increasing accumulation of body fat but

also, very likely, replacement of some of the muscle by fatty tissues.

It is also important to know the highest weight ever reached by the patient prior to his diabetes, for the weight at the time of the first observation is often not a reliable criterion. Many of these individuals may have lost twenty or thirty pounds before appearing for diagnosis of their condition. John (5) stresses this point in his statistical study of 2,970 diabetic persons on whom data were available as to maximum weight. He found that 62 per cent of the men and 69½ per cent of the women had been at some time 11 per cent or more overweight. Only 38 per cent of the men and 30½ per cent of the women were of normal weight or below throughout their lives.

It is a common observation that the incidence of diabetes is low where food is scarce, as was the case in some of the occupied countries during World War II. And it is not a mere coincidence that the United States, which is certainly the best fed and probably the most overfed nation in the world, also has the highest incidence of diabetes.

Just as obesity predisposes to the development of diabetes, so does continued overweight shorten the life expectancy of the diabetic individual. For men past age forty-five, the diabetic mortality is low for those of normal weight, doubles for those 5 to 14 per cent overweight, is four times as great for those 15 to 24 per cent overweight, and is ten times as great for those over 25 per cent overweight (6). The inference is that obesity accelerates the vascular degenerative changes to which the diabetic patient is particularly prone.

Although one can readily show the association of obesity and diabetes in about 80 per cent of adult cases, one cannot so readily explain the part played by overweight in producing the disease. Obviously obesity alone does not cause diabetes. Of all the fat people there are in this world, only a small percentage of the total ever develop it. Many factors undoubtedly operate to make the relationship obscure and of these, heredity is probably the most important.

White and Pincus (7) have shown that the diabetic tendency is inheritable in a mendelian recessive pattern. Its simultaneous occurrence in identical twins has been frequently noted and the disease has been found to occur five times as often in the blood relatives of people with diabetes as it does in the general population (8). In a recent follow-up study of 516 patients under age forty with diabetes of twenty-five or more years' duration, the incidence of hereditary diabetes was 44 per cent (9). Joslin suspects that a familial history of diabetes would be found for all diabetic patients if one only knew their ancestry.

Endocrine disturbances in other ductless glands, particularly the pituitary and adrenals and to a lesser extent the thyroid, further complicate the role played by obesity. Diseases commonly supposed to be caused by hyperfunction of these glands are frequently associated with hyperglycemia and glycosuria. More convincing evidence of the relationship between these glands and glucose metabolism is provided by the effects of their removal in experimental animals made diabetic by pancreatectomy. Houssay showed that hypophysectomy markedly reduced the severity of the diabetes, and similar effects were demonstrated by Long and Lukens (10) after adrenalectomy. Campbell and his associates (11) have been able to produce permanent diabetes in dogs by the administration of a highly purified growth hormone preparation of the anterior pituitary. These findings have led to the hypothesis that the anterior lobe contains a hormone which counteracts the insulin in some way, and that if this hormone is in great excess it may cause such activity in the islet cells that they are overworked and atrophy.

Dohan and Lukens (12) have been able to produce permanent diabetes in cats by the intraperitoneal injection of 20 per cent glucose. It is of interest that the caloric intake of these animals was equal to or less than that of normally fed cats and there was loss of body weight in all but one instance. It seemed that neither excess of calories nor increase in body mass were factors in the production of islet lesions in these experiments. Whether or not there is some agent

in addition to hyperglycemia, Luken's work suggests that an excessive functional demand on the pancreas leads to the breakdown of function and to anatomical changes of the islands.

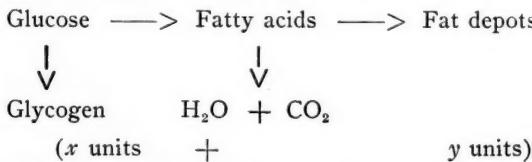
In summarizing this endocrine relationship, I think we may safely say that hyperglycemic disorders are caused either by underactivity of the pancreatic islets or by overactivity of the pituitary, adrenal, or thyroid glands.

Before considering the factor of overweight, I should like to review briefly some of the pertinent points in carbohydrate metabolism. The glucose of the body arises from several sources, the most important of which is the intestinal absorption of the products of carbohydrate digestion. The second, a process called glycogenolysis, is the production of glucose from glycogen under the influence of a specific enzyme, phosphatase, which is abundantly present in liver but not in muscle. A third source of glucose is termed glyconeogenesis, which means the formation of glucose from noncarbohydrate precursors such as protein and fat. It is not known to what extent in man each of these sources contributes to the total body glucose. However, in the rat, employing the technic of isotopic tracers, it was found that of the total glucose supply approximately two thirds was of dietary origin, about one third resulted from glyconeogenesis, and only about 3 per cent arose by glycogenolysis.

The above paragraph refers to the source of the glucose in the body. We shall now consider the fate of this glucose. Glucose is believed to be metabolically an entirely inert substance until it is changed to glucose-6-phosphate. This change is called phosphorylation, a process catalyzed by the ubiquitous enzyme, hexokinase. From there on, three chief pathways are open. A relatively small portion may be converted to glycogen and stored in the liver and muscles. Much the larger amount is converted to pyruvate. The greatest portion of carbohydrate energy is given by the burning of pyruvate over the tricarboxylic acid cycle to give, ultimately, carbon dioxide and water. A portion of pyruvate is utilized each day

in the synthesis of fatty acids. These, too, are burned to carbon dioxide and water. If, however, the calories of the diet exceed the caloric needs of the body, the excess of fatty acids is deposited in the body as neutral fat. Here again little is known of the extent of these several fates of glucose in man. Experimentally, it has been found that only about 3 per cent of the glucose ingested is converted each day to glycogen while about 30 per cent is consumed in the manufacture of fatty acids (13).

With this background, we are in a position to appreciate Long's (14) speculation on the relationship between obesity and diabetes. He assumed that the normal person, while maintaining a constant weight, requires  $x$  units of insulin to keep the following scheme in equilibrium, but when there is an excessive carbohydrate intake, the superfluous fatty acids are laid down in fat depots and for this an addition of  $y$  units of insulin is necessary:



In the obese person the chronic increased demand on the pancreas to supply  $x + y$  units of insulin might be expected to eventually overtax its capacity and thus bring on diabetes. Such diabetes is at first reversible by weight reduction, but there is a tendency toward its permanent establishment with increasing duration of the obesity. Obesity has been produced experimentally in Long's laboratory by hypothalamic lesions which lead to an uncontrollable appetite. Rats, which as a genus are characterized by a good reserve of islet function, develop typical diabetes when made obese in this way, and so do certain monkeys. Diabetic animals subsequently rendered obese by hypothalamic lesions show pronounced exacerbation of their diabetes.

Obviously this theory does not explain the development of diabetes in children, few of whom are overweight, or in the

habitually underweight adult. In these cases hereditary and endocrine factors are probably of paramount importance. It is logical to assume that a child who has inherited an inadequate pancreas may develop frank diabetes under the stress and strain of rapid growth resulting from an overactive pituitary gland.

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PRESIDENT YLVISAKER—Thank you, Dr. Trapnell. I hope we can hear again from you and from other younger men in our organization. I think all of you know Dr. Dillon from his previous appearances before this group and before the Medical Section of the American Life Convention. Dr. Dillon is one of the leading authorities on diabetes and a former president of the American Diabetes Association. I am sure you will be glad to hear him continue this discussion.

DR. EDWARD S. DILLON—Mr. President, Members of the Association: We have been considering why it is that overweights have a high mortality. This is considered to be due to three causes: atherosclerosis, diabetes, and hypertension.

Why do the diabetics have a high mortality? In the old days, of course, it was due primarily to coma, to severe

acidosis. That no longer is the leading cause. In fact, in many clinics it is rapidly disappearing and is almost no cause at all. However, in hospitals like the Philadelphia General Hospital where the patients are often brought in by ambulance or police patrol car, and usually twenty-four hours later than in other hospitals, we still lose cases from coma. This ought not to be so, and I hope the time will come when there will be no deaths from diabetic acidosis. Biogenic infections are still important but they have been very largely controlled by the advent of antibiotics.

Tuberculosis still accounts for some. In a recent study in Philadelphia where we reviewed 3,107 diabetics, we found that the frequency of tuberculosis in those cases was about double what it was in a large group of industrial workers.

Atherosclerosis and other degenerative lesions are, however, far and away our main problem. I should like to emphasize that in diabetic persons we have by far the largest group in the country in which atherosclerosis is early and severe. Why does atherosclerosis develop in diabetics? That is a question that I very much wish I could answer. We know that it does develop early. We know that it develops more rapidly. We know that it is in the coronary arteries and in the arteries of the lower extremities where it is most severe.

Those of us who have practiced the specialty of diabetes for some years and belong to the old conservative school led by Dr. Frederick M. Allen and Dr. Elliott P. Joslin feel very strongly that insofar as anything will prevent atherosclerosis it is the control of the diabetes. That word "control" has almost as many meanings as there are doctors in the world. Each one of us has perhaps a different conception of what constitutes control and what evidence is necessary to produce proof that the various criteria of control are being fulfilled.

In our clinic we have the following definition of control. The patient must be on a physiologic diet adequate to maintain his body weight at standard levels and no more—no

excess calories; the blood sugar every day, and all day every day, shall be between the levels of 80 and 180 mg. We believe that the blood sugar is likely to be within those levels if, first of all, the patient has no shocks and, second, if he has no sugar in the urine. In addition, he should have a fasting blood sugar of a desirable level for that particular patient.

Those are high standards which are not easy to fulfill. A patient has to be fairly constantly under observation and altogether cooperative before we may expect that he is being controlled as defined. There are certain brittle cases, as we call them, chiefly juveniles, in whom the blood sugar changes with enormous rapidity. The blood sugar might very well be 350 mg. fasting. If we give a fairly large dose of insulin, three hours later the blood sugar may be down to 50 mg. It changes with enormous speed. It is utterly impossible to keep those patients sugar-free and at the same time keep them out of shock. Anyone who attempts to keep such patients without sugar in the urine is flirting with danger, because it cannot be done.

Our philosophy of treatment with those cases, then, has to change somewhat. I like to give them as much insulin as I possibly can and still keep them out of shock.

Now, then, if we say that control of the diabetes is the chief important point in preventing atherosclerosis, what are we to believe about the physicians who treat their diabetics with free diets? They have a different set of standards as to what they consider constitutes good control. The results they get, to me, are quite confusing. Some of these gentlemen say that premature development of atherosclerosis is so constant as to constitute part and parcel of the disease of diabetes itself and that it is absolutely unpreventable. Then, again, there will be others who say that they have no more atherosclerosis in their patients than the rest of us do.

I do not know what the explanation is. I suppose we ought to get together and compare our patients, particularly the types of patients we are talking about. If we are talking about these very brittle diabetics, maybe we are on the same

ground. If we are talking about the very mild diabetics who can be controlled according to our standards by diet alone, then we are speaking an entirely different language.

I am sure that a very large percentage of the patients I have seen with gangrene which develops as a result of atherosclerosis of the arteries of the lower extremities have this in common: Usually their diabetes is relatively mild, and has been out of control over a long period of time.

As for fat in the diet, we have never felt that that was an important point. We believe the important points are: 1—control of the diabetes; and 2—control of body weight by giving no more calories than are necessary. Newburgh at the University of Michigan for many years, perhaps twenty-five, treated patients with high fat and low carbohydrate diets. Dr Kahn, who is professor of medicine and now in charge of the metabolic division, tells me that no accurate follow-up study has been made of these patients but that he is quite sure there is no more atherosclerosis in these patients of Dr. Newburgh's than in those who have been on diets of other types.

Random cholesterol studies of our patients made in the past have been exceedingly disappointing, both in satisfactory prognosis and as a guide for treatment. In the light of the newer studies it is quite obvious why that is so. The total cholesterol content itself is probably unimportant. We very much need a large quantity of new studies on patients who are sufficiently cooperative so that we have faith in what they are doing, and the type of studies where we take into consideration the phospholipid-total-cholesterol ratio which I believe, from the little experience I have had with it, to be exceedingly important.

PRESIDENT YLVISAKER—Thank you very much, Dr. Dillon and Dr. Trapnell.

We now come to the last paper on our program which I asked my associates to prepare. We have noted in our daily selection work that arterial hypertension is frequently present

in overweight applicants. Dr. Joyce Sheridan and Dr. John Peck have reviewed our material and have prepared a discussion on "Overweight as a Contributing Factor in the Development of Hypertension." Dr. Sheridan will present this discussion.

## OVERWEIGHT AS A CONTRIBUTING FACTOR IN THE DEVELOPMENT OF HYPERTENSION

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and*

JOHN McC. PECK, M. D., *Assistant Medical Director  
Fidelity Mutual Life Insurance Company  
Philadelphia, Pennsylvania*

Dr. Elliott P. Joslin (1) has commented that scales are to chronic disease what the thermometer is to acute illness. The extensive build mortality investigation which Dr. Dublin and Mr. Marks have brought before us today adds new emphasis to that comment. Far beyond its value for underwriting purposes in our work, this investigation serves as a new guide-post and challenge in the field of public health. It has once more amply demonstrated the excessive mortality that occurs among obese persons. The causes of death, as analyzed, help explain the channels through which the excess force of mortality, in obesity, operates. Most overcrowded of the avenues on which the obese move more hurriedly to death is that of arteriosclerosis—coronary, cerebral, or renal. And while it often fails to appear on the death certificate as the primary cause of death, hypertensive cardiovascular disease has a prominent share in this terminal picture.

Overweight and hypertension are of unique interest in that neither is a direct cause of death but either causes an increased death rate. In general, the greater the overweight, or the greater the hypertension, the higher the death rate will be. The effect of the two combined has unfortunately not been adequately studied in insurance mortality investigations. Overweight and hypertension have one feature in common: The excess deaths associated with either are mainly due to arteriosclerosis.

In some respects, it is odd that hypertension and overweight so often go hand in hand. When we think of persons predis-

posed to hypertension, we think of the hyperactive type—dynamic individuals with overactive sympathetic nervous systems, increased emotional tension and suppressed aggressiveness. By contrast, the popular and often the clinical impression of the obese person is that of the slow moving, plethoric, inactive type. About the only state of hyperactivity we associate with obesity is hyperactivity at the dinner table.

In spite of these popular concepts, repeated studies have demonstrated the frequency with which hypertension is obesity's companion. Thomson (2) reviewed the annual health records of 3,343 individuals employed by the Metropolitan Life Insurance Company. The average period of observation exceeded twelve years. He found that elevated diastolic blood pressure occurred most frequently among persons above average weight, those who weighed over 2.4 pounds per inch of height. Those of medium weight had a definitely lower incidence and those of light weight had the lowest incidence of all. At ages 35 to 44, diastolic hypertension was almost three times as common among those of heavy weight as among those of light weight. At ages over 45, diastolic hypertension was over twice as common among those of heavy weight as among those of light weight.

Robinson and Brucer (3) classified 3,658 persons into two groups: those with chest girth less than 50 per cent of height, the so-called "lineals," and those with chest girth 60 per cent or more of height, the so-called "laterals." Of the female "laterals," 27 per cent, and of the male "laterals," 22 per cent had systolic blood pressures above 140 mm. Only 2 per cent of the female "lineals" and 4 per cent of the male "lineals" had blood pressures above 140 mm. Elevated blood pressure was five to ten times as common among those of stocky build as among those of slender build.

This same influence of overweight as a predisposing factor in hypertension is evident among our employees. We have reviewed 3,529 examinations of 231 persons employed five years or more. Only 3 (2 per cent) of 163 employees of light or medium weight have had a diastolic blood pressure exceeding 90 at any time. This is in contrast to the 68 heavy weight

employees, one fourth of whom have had a diastolic blood pressure above 90 on one or more occasions. Hypertension has been over eight times as common among those who are above average weight. As in Thomson's (2) review, individuals weighing less than 2.0 pounds per inch of height have been considered as light weights; those from 2.0 to 2.4 pounds per inch, medium weights and those weighing over 2.4 pounds per inch of height, heavy weights.

We can observe this relationship from another angle. Of 231 persons employed for five years or more, one fourth (25.8 per cent) have had a blood pressure exceeding 140 systolic or 90 diastolic, or both, on one or more occasions. Of the 59 persons with that degree of intermittent or constant hypertension, 39 (66 per cent) have been overweight, that is, have weighed more than 2.4 pounds per inch of height. Of the 172 with no hypertension, only 49 (28.5 per cent) have been overweight. In other words, weight above average has been almost two and one-half times as common among the hypertensives as among the nonhypertensives.

Another daily reminder of the association between hypertension and obesity is the picture we see among examined applicants for insurance. It is both interesting and unusual that of 194 applicants whom the Fidelity Mutual Life Insurance Company last year found ineligible for standard insurance because of blood pressure, not one was a light weight. Over 70 per cent were of heavy weight.

Even more dramatic than this frequency with which hypertension and overweight are associated is the extent and rapidity with which blood pressure often drops when overweight persons are placed on a restricted diet. Dr. Ella Roberts (4) recently commented on seven hypertensives who had an average weight loss of 25 pounds during a three-month period of treatment directed to weight reduction. In all but one case blood pressure was reduced at the end of the three-month treatment period.

In our employee health program, we have repeatedly observed a striking blood pressure reduction following dietary

restriction instituted to correct obesity or for some other reason. N. A., for instance, entered employment in 1948 at age 23 with a weight of 219½ pounds and height of 73½ inches. Blood pressure on first three annual examinations varied from 120/90 to 128/80. A peak weight of 231 pounds was reached in November, 1950. By July, 1951, N. A.'s blood pressure had ascended to 164/116. He was placed on a 1,200 calorie diet. In two months his weight dropped to 205 pounds and his blood pressure gradually returned to normal levels, from 125/82 to 132/88. Sedentary life while studying for important examinations may have been a factor in N. A.'s weight gain. The attendant emotional stress may have been a precipitating factor in his hypertension. The hypertension, however, persisted after the emotional strain was removed. The hypertension disappeared during the period of dietetic restriction and weight reduction.

X. N. D. entered employment at age 48, in 1930, with height of 67 inches and weight of 167 pounds. His blood pressure was 175/90 on entry, with range from 152 to 175 systolic and 84 to 100 diastolic until 1949, when he developed diabetes. His weight was then 189 pounds. Modification of diet brought about prompt and complete control of his diabetes. He is one of the few cases we have seen in which a repeatedly demonstrated abnormal blood sugar tolerance has, under treatment, reverted to a consistently normal blood sugar tolerance. Of chief interest to the present discussion is the fact that X. N. D., who had frank hypertension for 19 years has now shown a consistently normal blood pressure during the two years of restricted diet. While limited, his diet is nicely balanced and entirely adequate to support normal weight and activity.

Brozek (5) and associates placed 34 normal young men on a daily intake of slightly under 1,600 calories. The average body weight for the group dropped about 24 per cent in a six-month period. Paralleling the decline in body weight were striking decreases in blood pressure. The mean systolic blood pressure for the entire group dropped almost 12 mm., from 106.5 mm. to 94.7 mm. The mean diastolic pressure of

the group dropped over 5 mm., from 69.9 mm. to 64.5 mm. Brozek's study demonstrates that even among persons whose blood pressure is quite normal, restricted diet brings about a reduction of blood pressure. It is of interest to note that sodium intake was not reduced by Brozek. He raises the question as to whether it is the low sodium level or merely the limited intake of an unsavory diet that is primarily responsible for the blood pressure reduction many patients exhibit under the Kempner regimen.

When we consider overweight as a contributing factor in the development of hypertension, we must not forget that overweight is primarily an index of overeating. Overeating, overweight and hypertension play allied roles in the chronic degenerative diseases of middle and later life, particularly atherosclerosis.

These limited observations, added to the many that others have made before, serve but a single purpose. They re-emphasize Dr. Dublin's profound comment that "the best means of reducing the incidence of degenerative diseases at middle and later life is by weight control." Armstrong, Dublin, Wheatley and Marks, at the meeting of the American Medical Association in Atlantic City last June, stressed anew the clear evidence that hypertension and degeneration of the heart and blood vessels are much more common in the obese than in others. "The time has come," they said, "for a comprehensive attack on obesity as a problem in public health."

The campaign for weight control, as a health measure, is one in which the insurance medical officer can readily participate. The dangers of obesity and the values of weight control can be stressed in employee health programs and in contact with agents, applicants, policyholders, examiners and attending physicians.

We believe that one of the values of the annual health audit can be its aid in checking the natural tendency to an unhealthy gain in weight with increasing age. Among Fidelity Mutual employees who have had such a health audit for five years or more, 60 per cent are now below their peak weight.

Weight control is a simple and important field of prophylactic medicine. The great campaign on which the Metropolitan Life Insurance Company has embarked, to educate patient and physician alike to the dangers of obesity, is one worthy of the cooperation and support of us all.

## REFERENCES

1. Joslin, E. P.: *Bost. Med. & Surg. Jl.* 186:833, 1922.
2. Thomson, K. Jefferson: *Some Observations On The Development And Course Of Hypertensive Vascular Disease*, *Proc. Med. Sec. A. L. C.* (June) 1950: 85-112.
3. Robinson, S. C. and Brucer, M.: *Arch. Int. Med.* 66:393, 1940.
4. Roberts, Ella: *The Treatment Of Obesity With Anorexigenic Drugs*, *Annals of Internal Medicine* 34:1324-1330, (June) 1951.
5. Brozek, Joseph, Chapman, C. B., and Keys, Ancel: *Drastic Food Restriction*, *J. A. M. A.* 137:1569-1574, (Aug. 28) 1948.

PRESIDENT YLVISAKER—Thank you, Dr. Sheridan.

Mr. Marks of the Metropolitan Life Insurance Company has contributed much to the Metropolitan studies, was along in presenting the exhibits at Atlantic City, and has helped us in preparing this part of our program. We shall appreciate your further comment, Mr. Marks.

MR. MARKS—Dr. Ylvisaker, Members of the Association: We have seen from so many points of view what obesity means, both to this business and to the nation's health. If there is one thing I would stress, it is that in the field of underwriting overweight has become not less of a problem but more of a problem.

In the life insurance business our ratings are, in a sense, all relative, and in terms of the position of overweights as against persons of lighter build, the situation of the overweight has deteriorated. Of course, overweights in common with other people have benefited from the advances in modern medicine but not to so great an extent. That underlies the change that has occurred in their relative position in terms of rating.

I might add that if we had concentrated our experience more in recent years we would have seen a picture somewhat

worse than we actually showed, because the most dramatic change in the reduction of infections in adult life has occurred since 1940, perhaps one or two years earlier. It began with the sulfa drugs which really did not come into wide use until about 1940. It continued with penicillin and all the other antibiotics that have come into use since. Consequently, on a current basis the situation among overweights—even moderate overweights—is even less favorable today than it was at the time of the beginning of our study. That is of extreme importance to us all as underwriters.

This new study has many other implications. We have given our experience on these substandard overweights. However, the material of the study is not as pure as we would like. As Dr. Bonnett has pointed out, there is still some screening to be done. If one takes unselected overweights, I think the picture for them is even worse than for our selected group. Again I would emphasize, as others have, that in our efforts to get people to reduce weight and to persist in weight reduction, we are performing a prime service to the public health as well as to our own business.

**PRESIDENT YLVISAKER**—Thank you, Mr. Marks. This completes the program for this year, and with it my year as president comes to an end. Before closing the meeting, I want to thank all the guest speakers who have contributed so much to a very excellent program.

I want also to thank all our members who have prepared formal papers, and who have otherwise entered into our discussions. I know how much work it is for both the guest speakers and our own members to prepare these discussions. It means work at night, work over the weekend, and interruption of flow of the regular work at the office.

I want to express my appreciation also to all the officers of our Association, the Executive Council, all our committees, and the committee chairmen for all their accomplishments during the year. I never realized before how much work our Association officers and committee members have to do.

I want to thank Dr. Kirkland for his help. The secretary has many responsibilities. The editor of our Transactions, Dr. Gudger, likewise has a tremendous task in editing all the papers and discussions for our book. I hope we can all help him by submitting our discussions to him as early as possible and in satisfactory form.

We also owe Dr. Dewis and his convention committee and the Hotel Statler staff our thanks for providing us with such excellent accommodations for our meeting.

It now becomes my pleasant duty to introduce to you your new officers. As you know, Dr. Kirkland has been re-elected secretary, and Dr. Gudger has been re-elected editor of the Transactions. The excellent work which both have done for us speaks for itself. Dr. Reiter is again your treasurer, and we all appreciate the splendid help he in this capacity has always given us.

Your new second vice president is Dr. R. C. Montgomery of the Manufacturers Life Insurance Company of Toronto; your new vice president is Dr. Earl C. Bonnett of the Metropolitan Life Insurance Company; and your new president is Dr. Linford H. Lee of the Pacific Mutual Life Insurance Company of Los Angeles. With these gentlemen directing our Association affairs, we can rest assured that we can look forward to a happy and successful year.

Dr. Lee, it is a pleasure for me to turn over this office to you. Your election shows that you have the respect and confidence of all the members of our organization, and I am glad to have the privilege of introducing you as our president for the coming year.

PRESIDENT-ELECT LINFORD H. LEE—Gentlemen, may I ask whether there is any more business to come before this meeting.

DR. EDWIN DEWIS—I would like to say that a vote of thanks should be extended to Dr. Ylvisaker for the excellent program that he arranged and for the splendid way in which he has conducted the meeting.

PRESIDENT-ELECT LEE—I would suggest that we all show our appreciation by standing.

Gentlemen, I am sure that I need not go into detail about my emotional reactions at this time. To be elected president of this old and famous organization is a very great honor. Little did I think when I attended my first meeting in 1934 that I would ever receive this honor. At that time, Dr. Frazer of the New York Life Insurance Company was president. He very kindly asked me to read a little paper on attending physicians' statements. Many complimented me, and I felt pretty good about it. Dr. Frazer said that he had invited me to read a paper in spite of my youth and inexperience because he feared my superior would not allow me to attend otherwise. So, he rather took me down a bit.

Our meetings have always been characterized by good fellowship and an exceptionally high standard of scientific program, as demonstrated here this year. The work of Dr. Ylvisaker and those who have preceded him and me has been of such high caliber that it becomes very difficult to follow them. However, with the help of you gentlemen on whom I shall call, and the other officers of the Association, I assure you that we are not going to leave any stones unturned to carry on in the splendid tradition that is characteristic of the history of this fine organization.

It is my great pleasure to be able to say that I have the favorable reaction of your Executive Council to plan for our next meeting to be held in California. The place is Los Angeles, and the date is the week of October 20, 1952. Many of you have already told me that you are planning to be there, combining the trip to the Convention with business and visiting your agencies or your examiners, as is done by members from the Pacific coast who come to the meetings here. In addition, may I suggest that you also consider the possibility of combining a trip to the meeting out there with your vacation, bringing along members of your family.

Our main effort will be directed toward the building of a scientific program that will meet with your approval. How-

ever, we shall definitely have some plans for the comfort and pleasure of you and those who may accompany you. I am sure you will have an enjoyable visit. I hope that I shall have the pleasure of greeting you all next October in California.

Thank you all very much for the confidence you have placed in me by electing me to the office of President of the Association.

Since the secretary informs me that there is no more business to come before this Convention, I declare it closed.

The following doctors were present at some time during the sessions:

C. B. Ahlefeld	B. R. Comeau	O. G. Goldkamp
Joseph Altman	W. P. Constable	R. A. Goodell
K. W. Anderson	J. L. Cook	George Goodkin
W. B. Aten	H. K. Crutcher	H. W. Goos
D. R. Auten	B. A. Dawber	J. K. Gordon
Bernard Baillargeon	H. D. Delamere	C. D. Gossage
Max Bakalinsky	E. J. Dewees	A. S. Graham
N. J. Barker	E. G. Dewis	Ghent Graves
G. P. Barnett	F. R. Dieuaide	C. J. M. Grisdale
C. C. Beach	E. S. Dillon	R. S. Gubner
J. R. Beard	H. W. Dingman	J. R. Gudger
E. W. Beckwith	J. P. Donelan	E. Y. Hall
R. A. Behrman	G. D. Dorman	Llewellyn Hall
M. F. Bell	R. L. Dross	F. T. Hallam
M. B. Bender	L. I. Dublin	G. W. Halpenny
W. H. Bennett	F. P. Duchesneau	V. G. Hammond
W. R. Bishop	T. C. Dunlop	O. E. Hanes
J. E. Boland	L. B. Dunn	A. H. Hansen
William Bolt	T. M. Durant	Frank Harnden
E. C. Bonnett	W. W. Eakin	L. E. Hathaway, Jr.
M. T. Boss	L. H. Earle, Jr.	H. L. Hauge
K. F. Brandon	T. M. Ebers	W. C. Hausheer
H. J. Brekke	Paul Engle	H. M. Hawkins
A. W. Bromer	A. H. Faber	P. S. Hench
F. R. Brown	R. B. Failey	O. C. Hendrix
H. B. Brown	J. G. Falconer	E. V. Higgins
Leslie Brown	R. K. Farnham	H. E. Hilleboe
R. F. Buchan	H. H. Fellows	W. L. Hilliard
E. R. Bush	R. M. Filson	E. C. Hillman, Jr.
B. F. Byrd	R. W. Finegan	D. W. Hoare
J. T. Cabaniss	P. M. L. Forsberg	J. C. Horan
W. R. Calderwood	E. M. Freeland	E. G. Howe
E. J. Campbell	C. E. Fronk	T. B. Hoxie
P. E. Carlisle	I. K. Gardner	J. L. Humphreys
D. W. Carter	D. S. Garner	Arthur Hunter
J. P. Chapman	J. H. Geddes	J. J. Hutchinson
P. H. Charlton	W. M. Gentner	J. R. B. Hutchinson
R. B. Cleveland	E. E. Getman	F. J. Ingelfinger
N. B. Cole	R. T. Gilchrist	A. S. Irving
G. R. Collyer		J. G. Irving

## SIXTIETH ANNUAL MEETING

A. O. Jimenis	R. W. Mann	N. E. Ruud
A. E. Johann	H. M. Marvin	M. T. Ryman
J. W. Johnson, Jr.	F. A. L. Mathewson	K. F. Schaefer
	L. K. Meredith	L. P. Schroeder
J. R. Karns	E. B. Milam	B. T. D. Schwarz
V. L. Karren	M. B. Miller	W. H. Scions
E. A. Keenleyside	E. S. Mills	D. L. Selby
N. R. Kelley	J. T. Montgomery	E. F. Sheldon
E. F. Kerby	R. C. Montgomery	J. T. Sheridan
H. B. Kidd	S. R. Moore	J. F. Shortsleeve
C. E. Kiessling	J. R. E. Morden	R. R. Simmons
D. G. Kilgore	J. P. Moss	A. M. Sison
Richard King	C. V. Mulligan	Isaac Sossnitz
C. T. Kirchmaier	S. A. Narins	C. G. Spivey
H. B. Kirkland	R. M. Nay	F. L. Springer
	R. A. Nelson	H. F. Starr
G. C. LaBelle	A. J. Oberlander	F. R. Stearns
W. C. Lamb	Herbert Old	J. B. Steele
Phillips Lambkin	M. I. Olsen	D. F. R. Steuart
P. H. Langner, Jr.		F. M. Stites
H. F. Laramore	C. B. Parker	L. G. Sykes
A. L. Larson	A. E. Parks	B. C. Syverson
I. C. Lawler	J. S. Pearson	L. J. Tedesco
L. H. Lee	J. M. Peck	G. F. Tegtmeyer
E. P. Leeper	D. S. Pepper	R. W. Thayer
H. R. Leffingwell	H. M. Pequegnat	K. J. Thomson
W. R. Leute, Jr.	C. A. Peters	W. B. Thornton
J. C. Lindner	O. H. Peterson	A. R. Tormey
J. M. Livingston	J. C. Pierson	G. D. Townshend
G. W. Lougheed	Cullen Pitt	J. M. Trapnell, Jr.
G. J. Lunz	R. W. Pratt	F. D. Truax
	M. A. Puzak	H. E. Ungerleider
F. M. McChesney	O. S. Randall	B. W. Vale
R. R. McCormack	J. H. Ready	Euen Van Kleeck
H. M. McCue	Rezin Reagan	A. E. Venables
William MacDonald	C. L. Reeder	Vernon Van Zandt
A. J. McGanity	E. A. Reiman	
F. J. McGurl	P. V. Reinartz	P. C. Waldo
T. J. McGurl	W. A. Reiter	R. V. Ward
George McLean	W. M. Reynolds	C. F. Warren
L. L. McLellan	G. P. Robb	R. L. Weaver
W. J. McNamara	D. C. Roberts	Jefferson Weed
Charles Maertz	H. B. Rollins	W. L. Weeden
H. R. Magee	T. F. Ross	G. M. Wheatley
S. J. N. Magwood		
John Malgieri		

MEMBERS PRESENT

303

E. V. Wiedman	A. C. Wilson	R. W. Zinkann
J. A. Wilhelm		A. F. Zipf
A. A. Willander	L. S. Ylvisaker	
E. S. Williams	G. G. Young	
R. L. Willis		

Also present were:

Theodore Ake	H. H. Marks	A. E. Thyselius
James Andrews, Jr.	A. P. Morton	A. C. Webster
Edward King	B. S. Pauley	P. V. Wells
G. C. Kingsley	O. G. Sherman	J. C. Wilberding
Miss A. M. Lyle	W. M. Stufflebeam	

Total attendance at all sessions, 272.

## **In Memoriam**

### **Deceased since Fifty-Ninth Annual Meeting**

Thomas D. Archibald, M. D.  
North American Life Assurance Company  
Died September 2, 1951

Cecil C. Birchard, M. D.  
Sun Life Assurance Company of Canada  
Died July 30, 1951

Parker M. Cort, M. D.  
Aetna Life Insurance Company  
Died September 27, 1951

William G. Hyde, M. D.  
Northwestern Mutual Life Insurance Company  
Died March 27, 1951

H. Clive McAlister, M. D.  
Lincoln National Life Insurance Company  
Died April 28, 1951

Frederick W. McSorley, M. D.  
Equitable Life Assurance Society of the United  
States  
Died March 24, 1951

Wallace R. Richardson, M. D.  
National Equity Life Insurance Company  
Died November 15, 1950

Albert Seaton, M. D.  
American United Life Insurance Company  
Died November 12, 1950

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE  
INSURANCE MEDICAL DIRECTORS OF AMERICA

Charles B. Ahlefeld, M. D.	Business Men's, Kansas City, Mo.
George E. Allen, M. D.	National, Montpelier, Vt.
Joseph Altman, M. D.	Companion Life, New York City
Henry H. Amsden, M. D.	United Life and Accident, Concord, N. H.
E. A. Anderson, M. D.	Modern Woodmen, Rock Island, Ill.
Frank R. Anderson, M. D.	Pacific Mutual, Los Angeles, Calif.
Karl W. Anderson, M. D.	Northwestern National, Minneapolis, Minn.
Perry A. Anderson, M. D.	Rockford Life, Rockford, Ill.
Robert L. Anderson, Jr., M. D.	Reliance, Pittsburgh, Pa.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
William B. Aten, M. D.	Security Mutual, Binghamton, N. Y.
Donald R. Auten, M. D.	New York Life, New York City
J. Albert Avrack, M. D.	United States Life, New York City

Bernard Baillargeon, M. D.	Alliance Nationale, Montreal, Canada
G. Holbrook Barber, M. D.	Manhattan, New York City
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
Gordon P. Barnett, M. D.	Kansas City Life, Kansas City, Mo.
Charles M. Barrett, M. D.	Western and Southern, Cincinnati, Ohio

Daniel S. Baughman, M. D.	Security Life and Accident, Denver, Colo.
Earl G. Baxter, M. D.	Franklin, Springfield, Ill.
Carroll C. Beach, M. D.	State Mutual, Worcester, Mass.
J. Randolph Beard, M. D.	Mutual Benefit, Newark, N. J.
Edgar W. Beckwith, M. D.	Equitable Life Assurance, New York City
James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.
Roland A. Behrman, M. D.	John Hancock Mutual, Boston, Mass.
J. V. Bell, M. D.	National Fidelity, Kansas City, Mo.
Murray F. Bell, M. D.	New York Life, New York City
Maurice B. Bender, M. D.	Guardian, New York City
Robert A. Benson, M. D.	Metropolitan, New York City
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
C. Coleman Berwick, M. D.	Metropolitan, New York City
Francis P. Bicknell, M. D.	State Mutual, Worcester, Mass.
J. Rozier Biggs, M. D.	Peoples, Washington, D. C.
B. Cosby Bird, M. D.	Preferred, Montgomery, Ala.
William R. Bishop, M. D.	Provident Life Acc., Chattanooga, Tenn.
Norman R. Blatherwick, M. D.	Metropolitan, New York City
John E. Boland, M. D.	Country, Chicago, Ill.
William Bolt, M. D.	New York Life, New York City
John M. Bond, M. D.	Northwestern Mutual, Milwaukee, Wis.
Earl C. Bonnett, M. D.	Metropolitan, New York City
M. Theodore Boss, M. D.	Home Friendly, Baltimore, Md.

## LIST OF MEMBERS

307

J. Thornley Bowman, M. D.	London Life, London, Canada
Ernest L. Boylen, M. D.	Standard, Portland, Ore.
Kenneth F. Brandon, M. D.	Aetna, Hartford, Conn.
David J. Breithaupt, M. D.	Manufacturers, Toronto, Canada
Albert W. Bromer, M. D.	Metropolitan, New York City
C. Frank Brown, M. D.	Southwestern, Dallas, Tex.
Frederick R. Brown, M. D.	New England Mutual, Boston, Mass.
Howard B. Brown, M. D.	Massachusetts Mutual, Springfield, Mass.
Leslie Brown, M. D.	Equitable Life Assurance, New York City
Ronald F. Buchan, M. D.	Prudential, Newark, N. J.
Earl R. Bush, M. D.	Western and Southern, Cincinnati, Ohio
Benjamin F. Byrd, M. D.	National Life & Accident, Nashville, Tenn.
Joseph T. Cabaniss, M. D.	Travelers, Hartford, Conn.
Edward J. Campbell, M. D.	New York Life, New York City
Hugh B. Campbell, M. D.	Phoenix Mutual, Hartford, Conn.
Raymond L. Candage, M. D.	John Hancock Mutual, Boston, Mass.
Paul E. Carlisle, M. D.	Prudential, Los Angeles, Calif.
Laurence D. Chapin, M. D.	Massachusetts Mutual, Springfield, Mass.
John P. Chapman, M. D.	Pennsylvania Life, Health & Accident, Philadelphia, Pa.
Paul H. Charlton, M. D.	Midland Mutual, Columbus, Ohio
Edmund D. Chesebro, M. D.	Puritan, Providence, R. I.
Harry E. Christensen, M. D.	Union Mutual, Portland, Maine

## SIXTIETH ANNUAL MEETING

Robert B. Cleveland, M. D.	Equitable Life Assurance, New York City
Joseph C. Clifford, M. D.	Aetna, Hartford, Conn.
Milton H. Clifford, M. D.	New England Mutual, Boston, Mass.
Harry A. Cochran, Jr., M. D.	Reliance, Pittsburgh, Pa.
Norman B. Cole, M. D.	Baltimore Life, Baltimore, Md.
Irwin E. Colgin, M. D.	Texas Life, Waco, Tex.
G. R. Collyer, M. D.	London Life, London, Canada
Harry L. Colombo, M. D.	National Life, Montpelier, Vt.
Frederick R. Congdon, M. D.	Berkshire, Pittsfield, Mass.
J. Lindsay Cook, M. D.	Pilot, Greensboro, N. C.
W. Pepper Constable, M. D.	Mutual, New York City
Neil L. Criss, M. D.	United Benefit, Omaha, Neb.
Howard K. Crutcher, M. D.	United Fidelity, Dallas, Tex.
Khurshed J. J. Cursetji, M. D.	Oriental Government Security Life, Bombay, India

Bryan A. Dawber, M. D.	Penn Mutual, Philadelphia, Pa.
John S. Delahaye, M. D.	Empire Life, Kingston, Canada
Harold D. Delamere, M. D.	Crown, Toronto, Canada
Aniceto Del Rio, M. D.	La Nacional, Mexico City, Mexico
Ernest J. Dewees, M. D.	Provident Mutual, Philadelphia, Pa.
Earle T. Dewey, M. D.	Metropolitan, New York City
Edwin G. Dewis, M. D.	Prudential, Newark, N. J.
Edward S. Dillon, M. D.	Penn Mutual, Philadelphia, Pa.

## LIST OF MEMBERS

309

Harry W. Dingman, M. D.	Continental Assurance, Chicago, Ill.
Nathaniel P. Doak, M. D.	Great Southern, Houston, Tex.
Albert H. Domm, M. D.	Prudential, Los Angeles, Calif.
James P. Donelan, M. D.	Guarantee Mutual, Omaha, Neb.
Gerald D. Dorman, M. D.	New York Life, New York City
James T. Downs, Jr., M. D.	Fidelity Union, Dallas, Tex.
Raymond L. Dross, M. D.	Prudential, Newark, N. J.
Thomas C. Dunlop, M. D.	Manufacturers, Toronto, Canada
Louis B. Dunn, M. D.	Postal, New York City
William W. Eakin, M. D.	Standard, Montreal, Canada
Lyon H. Earle, Jr., M. D.	Connecticut General, Hartford, Conn.
Theodore M. Ebers, M. D.	Connecticut Mutual, Hartford, Conn.
H. Glenn Ebersole, M. D.	Illinois Bankers, Monmouth, Ill.
Laurence B. Ellis, M. D.	Boston Mutual, Boston, Mass.
Jack A. End, M. D.	Northwestern Mutual, Milwaukee, Wis.
John L. Evans, M. D.	Farmers & Bankers, Wichita, Kan.
Albert H. Faber, M. D.	New York Life, New York City
J. Gilbert Falconer, M. D.	North American, Toronto, Canada
Raymond K. Farnham, M. D.	Metropolitan, New York City
Haynes H. Fellows, M. D.	Metropolitan, New York City
William S. Fewell, M. D.	Liberty, Greenville, S. C.
Ralph M. Filson, M. D.	Travelers, Hartford, Conn.

Rexford W. Finegan, M. D.	Metropolitan, New York City
Frederick Fink, M. D.	Franklin, Springfield, Ill.
Harry E. Flansburg, M. D.	Bankers, Lincoln, Neb.
James G. Forgerson, M. D.	Massachusetts Mutual, Springfield, Mass.
Philip M. L. Forsberg, M. D.	United Life and Accident, Concord, N. H.
Garth E. Fort, M. D.	National Life & Accident, Nashville, Tenn.
John M. Foster, M. D.	Capitol, Denver, Colo.
Edward M. Freeland, M. D.	New York Life, New York City
Clarence E. Fronk, M. D.	Hawaiian Life, Honolulu, T. H.
Harold M. Frost, M. D.	New England Mutual, Boston, Mass.
F. Irving Ganot, M. D.	Prudential, Newark, N. J.
I. Kenneth Gardner, M. D.	Reliance, Pittsburgh, Pa.
David S. Garner, M. D.	Shenandoah, Roanoke, Va.
J. H. Geddes, M. D.	Northern, London, Canada
John T. Geiger, M. D.	Metropolitan, New York City
Leo Gelfand, M. D.	Constitution Life, Los Angeles, Calif.
William M. Gentner, M. D.	Continental American, Wilmington, Del.
Edson E. Getman, M. D.	New York Life, New York City
John M. Gilchrist, M. D.	Monarch, Springfield, Mass.
Ralph T. Gilchrist, M. D.	Northwestern Mutual, Milwaukee, Wis.
Edgar G. Givhan, Jr., M. D.	Protective, Birmingham, Ala.
Otto G. Goldkamp, M. D.	Connecticut General, Hartford, Conn.

## LIST OF MEMBERS

311

Robert A. Goodell, M. D.	Phoenix Mutual, Hartford, Conn.
George Goodkin, M. D.	Equitable Life Assurance, New York City
Harry W. Goos, M. D.	Home, Philadelphia, Pa.
J. Keith Gordon, M. D.	Sun, Montreal, Canada
Charles D. Gossage, M. D.	Confederation, Toronto, Canada
Angus S. Graham, M. D.	London Life, London, Canada
George M. Graham, M. D.	Lincoln National, Fort Wayne, Ind.
Albert E. Gras, M. D.	Prudential, Newark, N. J.
Ghent Graves, M. D.	American General, Houston, Tex.
Marvin L. Graves, M. D.	American General, Houston, Tex.
Harris M. Gray, M. D.	Manufacturers, Toronto, Canada
Floyd M. Green, M. D.	Columbus Mutual, Columbus, Ohio
George E. Greenway, M. D.	Western Life Assurance, Hamilton, Canada
C. J. M. Grisdale, M. D.	Connecticut General, Hartford, Conn.
Frederick O. Gronvold, M. D.	Pioneer Mutual, Fargo, N. D.
Richard S. Gubner, M. D.	Equitable Life Assurance, New York City
James R. Gudger, M. D.	Mutual, New York City
Llewellyn Hall, M. D.	Phoenix Mutual, Hartford, Conn.
F. Tulley Hallam, M. D.	State, Indianapolis, Ind.
John H. Halliday, M. D.	Australian Mutual, Sydney Australia
Gerald W. Halpenny, M. D.	Royal, Montreal, Canada
Vincent G. Hammond, M. D.	Security Mutual, Binghamton, N. Y.

Ottis E. Hanes, M. D.	Life Ins. Co. of Ga., Atlanta, Ga.
John A. A. Harcourt, M. D.	Toronto Mutual, Toronto, Canada
Charles P. Hardwicke, M. D.	Western Reserve, Austin, Tex.
Frank Harnden, M. D.	Berkshire, Pittsfield, Mass.
Garland M. Harwood, M. D.	Life Insurance Co. of Virginia, Richmond, Va.
Louis E. Hathaway, Jr., M. D.	Monarch, Springfield, Mass.
Howard L. Hauge, M. D.	New York Life, New York City
Walter C. Hausheer, M. D.	Prudential, Newark, N. J.
Harry M. Hawkins, M. D.	Old Line, Milwaukee, Wis.
Thomas L. Hawkins, M. D.	Western, Helena, Mont.
Joseph K. P. Hawks, M. D.	State Farm, Bloomington, Ill.
J. Harry Hayes, M. D.	Union, Little Rock, Ark.
Olin C. Hendrix, M. D.	New England Mutual, Boston, Mass.
Ivan C. Heron, M. D.	West Coast, San Francisco, Calif.
William D. Hickerson, M. D.	Union Central, Cincinnati, Ohio
Eugene V. Higgins, M. D.	North American Reassurance, New York City
William L. Hilliard, M. D.	Equitable, Waterloo, Canada
Ernest C. Hillman, Jr., M. D.	Mutual Benefit, Newark, N. J.
Daniel W. Hoare, M. D.	Penn Mutual, Philadelphia, Pa.
Ira E. Hoffman, M. D.	Washington National, Evanston, Ill.
Joseph C. Horan, M. D.	Metropolitan, New York City
Arnold B. Houston, M. D.	Monarch, Winnipeg, Canada
Edward G. Howe, M. D.	Prudential, Newark, N. J.

## LIST OF MEMBERS

313

Thomas B. Hoxie, M. D.	New York Life, New York City
Henry W. Hudson, M. D.	Loyal Protective, Boston, Mass.
John L. Humphreys, M. D.	Reliance, Pittsburgh, Pa.
James H. Humphries, M. D.	Home, New York City
J. Edward Hunsinger, M. D.	Republic Nat'l, Dallas, Tex.
Benjamin L. Huntington, M. D.	John Hancock Mutual, Boston, Mass.
Samuel W. Hurdle, M. D.	Security Life & Trust, Winston-Salem, N. C.
John J. Hutchinson, M. D.	New York Life, New York City
J. Raymond B. Hutchinson, M. D.	Acacia Mutual, Washington, D. C.
Albert S. Irving, M. D.	Commonwealth, Louisville, Ky.
J. Grant Irving, M. D.	Aetna, Hartford, Conn.
Samuel Jagoda, M. D.	State Reserve, Fort Worth, Tex.
Albert O. Jimenis, M. D.	Metropolitan, New York City
Albert E. Johann, M. D.	Bankers, Des Moines, Iowa
Hubert R. John, M. D.	Maccabees, Detroit, Mich.
Joseph W. Johnson, Jr., M. D.	Interstate Life and Accident, Chattanooga, Tenn.
Alfred Kahn, Jr., M. D.	National Equity, Little Rock, Ark.
Victor L. Karren, M. D.	Home, New York City
Edward A. Keenleyside, M. D.	Prudential, Toronto, Canada
Charles H. Kelley, M. D.	Columbian National, Boston, Mass.

## SIXTIETH ANNUAL MEETING

Newell R. Kelley, M. D.	Phoenix Mutual, Hartford, Conn.
Herbert B. Kennedy, M. D.	Woodmen of the World, Omaha, Neb.
Harry B. Kidd, M. D.	Metropolitan, New York City
Charles E. Kiessling, M. D.	Prudential, Newark, N. J.
Donald G. Kilgore, M. D.	Republic National, Dallas, Tex.
Ray E. King, M. D.	Bankers, Des Moines, Iowa
Carl T. Kirchmaier, M. D.	Life & Casualty, Nashville, Tenn.
Henry B. Kirkland, M. D.	Prudential, Newark, N. J.
Edward Kuck, M. D.	Union Central, Cincinnati, Ohio
Earl J. Kuenster, M. D.	Paul Revere, Worcester, Mass.
Paul Kurzweg, Jr., M. D.	All American Assurance, Lafayette, La.
Walter C. Lamb, M. D.	Equitable Life Assurance, New York City
Phillips Lambkin, M. D.	Guardian, New York City
Paul H. Langner, Jr., M. D.	Provident Mutual, Philadelphia, Pa.
Louis G. LaPointe, M. D.	Manhattan Life, New York City
H. Franklyn Laramore, M. D.	Connecticut Mutual, Hartford, Conn.
Albert L. Larson, M. D.	Travelers, Hartford, Conn.
Ivan C. Lawler, M. D.	New York Life, New York City
Linford H. Lee, M. D.	Pacific Mutual, Los Angeles, Calif.
Edward P. Leeper, M. D.	Praetorians, Dallas, Tex.
Harold R. Leffingwell, M. D.	Paul Revere, Worcester, Mass.
William R. Leute, Jr., M. D.	Penn Mutual, Philadelphia, Pa.

T. Herbert Lewis, M. D.	Western States, Fargo, N. D.
Janus C. Lindner, M. D.	Prudential, Newark, N. J.
Everett H. Lindstrom, M. D.	Western, Helena, Mont.
James A. Livingston, M. D.	Liberty National, Birmingham, Ala.
John M. Livingston, M. D.	Mutual, Waterloo, Canada
Gladstone W. Lougheed, M. D.	Confederation, Toronto, Canada
Cabot Lull, M. D.	American, Birmingham, Ala.
Gerald J. Lunz, M. D.	Knights of Columbus, New Haven, Conn.

Frank M. McChesney, M. D.	Equitable, Washington, D. C.
William J. McConnell, M. D.	Metropolitan, New York City
George McCreight, M. D.	Bankers, Des Moines, Iowa
Howard M. McCue, Jr., M. D.	Life Insurance Co. of Virginia, Richmond, Va.
William MacDonald, M. D.	Teachers Insurance & Annuity Association, New York City
Arthur J. McGanity, M. D.	Dominion, Waterloo, Canada
Frank J. McGurl, M. D.	Prudential, Newark, N. J.
William G. McLaughry, M. D.	Protected Home Circle, Sharon, Pa.
George McLean, M. D.	Sun, Baltimore, Md.
Lawrence L. McLellan, M. D.	Provident Mutual, Philadelphia, Pa.
Ralph E. McLochlin, M. D.	National Old Line, Little Rock, Ark.
William J. McNamara, M. D.	Equitable Life Assurance, New York City
Charles Maertz, M. D.	Union Central, Cincinnati, Ohio

## SIXTIETH ANNUAL MEETING

Charles D. Magee, M. D.	Missouri Insurance Company, St. Louis, Mo.
S. J. Newton Magwood, M. D.	Continental, Toronto, Canada
John Malgieri, M. D.	New York Life, New York City
Robert W. Mann, M. D.	Imperial, Toronto, Canada
Francis A. L. Mathewson, M. D.	Great-West, Winnipeg, Canada
Loren K. Meredith, M. D.	National, Des Moines, Iowa
Ignacio Mesa, M. D.	"La Latino-Americana", Mexico City, Mexico
Ernest B. Milan, M. D.	Peninsular, Jacksonville, Fla.
Lloyd C. Miller, M. D.	National Life & Accident, Nashville, Tenn.
Edward S. Mills, M. D.	Prudential Assurance, Montreal, Canada
Eugene Montgomery, M. D.	North American, Toronto, Canada
James T. Montgomery, M. D.	North American Reassurance, New York City
Richard C. Montgomery, M. D.	Manufacturers, Toronto, Canada
John F. Moore, Jr., M. D.	Mutual, New York City
J. R. E. Morden, M. D.	Massachusetts Mutual, Springfield, Mass.
Reuben A. Moser, M. D.	American Reserve, Omaha Neb.
J. Palmer Moss, M. D.	Columbian Mutual, Memphis, Tenn.
Bernard Mount, M. D.	All States, Montgomery, Ala.
Elmer B. Mountain, M. D.	American Mutual, Des Moines, Iowa
Clifford V. Mulligan, M. D.	T. Eaton, Toronto, Canada
Frederick D. Munroe, M. D.	Fidelity, Regina, Canada
George H. Murphy, M. D.	Maritime, Halifax, Canada

## LIST OF MEMBERS

317

Sidney A. Narins, M. D.	Mutual, New York City
Richard M. Nay, M. D.	Indianapolis Life, Indianapolis, Ind.
Mather H. Neill, M. D.	Aetna, Hartford, Conn.
Clive P. Neilson, M. D.	Sovereign Life, Winnipeg, Canada
Richard A. Nelson, M. D.	Prudential, Newark, N. J.
Charles F. Nichols, M. D.	Penn Mutual, Philadelphia, Pa.
John B. Nichols, M. D.	Acacia Mutual, Washington, D. C.
Richard E. Nicholson, M. D.	Connecticut Mutual, Hartford, Conn.
E. Clark Noble, M. D.	National, Toronto, Canada
Andrew J. Oberlander, M. D.	National, Montpelier, Vt.
William L. O'Connell, M. D.	Union Labor, New York City
Martin I. Olsen, M. D.	Central, Des Moines, Iowa
William F. H. O'Neill, M. D.	Great-West, Winnipeg, Canada
Wilbert C. Page, M. D.	Prudential, Newark, N. J.
Charles B. Parker, M. D.	Independent Order of Foresters, Toronto, Canada
Arthur E. Parks, M. D.	Canada Life, Toronto, Canada
John S. Pearson, M. D.	John Hancock Mutual, Boston, Mass.
John M. Peck, M. D.	Fidelity Mutual, Philadelphia, Pa.
D. Sergeant Pepper, M. D.	Provident Mutual, Philadelphia, Pa.
Homer M. Pequegnat, M. D.	Dominion, Waterloo, Canada
Gilberto S. Pesquera, M. D.	Metropolitan, New York City

## SIXTIETH ANNUAL MEETING

Charles A. Peters, M. D.	Prudential Assurance, Montreal, Canada
Cullen Pitt, M. D.	Atlantic, Richmond, Va.
Roscoe W. Pratt, M. D.	New York Life, New York City
William O. Purdy, M. D.	Equitable, Des Moines, Iowa
Michael A. Puzak, M. D.	Peoples, Washington, D. C.
Louis A. Pyle, M. D.	Colonial, East Orange, N. J.
Edwin J. Quinn, M. D.	Mutual, New York City
O. Samuel Randall, M. D.	Midland National, Watertown, S. D.
Paul M. Rattan, M. D.	Great National, Dallas, Tex.
James H. Ready, M. D.	General American, St. Louis, Mo.
Rezin Reagan, M. D.	Policyholder's National, Sioux Falls, S. D.
Clifton L. Reeder, M. D.	Continental Assurance, Chicago, Ill.
Earl A. Reiman, M. D.	State Mutual, Worcester, Mass.
Paul V. Reinartz, M. D.	Prudential, Newark, N. J.
Walter A. Reiter, M. D.	Mutual Benefit, Newark, N. J.
Whitman M. Reynolds, M. D.	Equitable Life Assurance, New York City
H. Guy Riche, M. D.	Guaranty Income, Baton Rouge, La.
Donald F. Rikkers, M. D.	Northwestern Mutual, Milwaukee, Wis.
George P. Robb, M. D.	Metropolitan, New York City
David C. Roberts, M. D.	Guardian, New York City
Albert J. Robinson, M. D.	Connecticut General, Hartford, Conn.
Claude A. Robison, M. D.	Peoples, Frankfort, Ind.

## LIST OF MEMBERS

319

Henry B. Rollins, M. D.	Connecticut Mutual, Hartford, Conn.
Gordon Ross, M. D.	Massachusetts Mutual, Springfield, Mass.
John G. Ross, M. D.	Mutual, Waterloo, Canada
Thomas F. Ross, M. D.	Ohio State, Columbus, Ohio
Edward W. Rowe, M. D.	Midwest, Lincoln, Neb.
William W. Rucks, M. D.	Home State, Oklahoma City, Okla.
Merlin T. Ryman, M. D.	Mutual Benefit, Newark, N. J.
 Dan Y. Sage, M. D.	 Southern, Atlanta, Ga.
Joe H. Sanderlin, M. D.	Pyramid, Little Rock, Ark.
Raymond C. Scannell, M. D.	Security Life and Accident, Denver, Colo.
Kenneth F. Schaefer, M. D.	Prudential, Newark, N. J.
Berthold T. D. Schwarz, M. D.	Bankers National, Montclair, N. J.
William H. Scoins, M. D.	Lincoln National, Ft. Wayne, Ind.
Robert J. Scott, M. D.	Michigan Life, Detroit, Mich.
Alfred F. Seibert, M. D.	Travelers, Hartford, Conn.
David L. Selby, M. D.	Imperial, Toronto, Canada
Thomas S. Sexton, M. D.	Massachusetts Mutual, Springfield, Mass.
Hall Shannon, M. D.	Southland, Dallas, Tex.
Elroy F. Sheldon, M. D.	Occidental, Los Angeles, Calif.
Joyce T. Sheridan, M. D.	Fidelity Mutual, Philadelphia, Pa.
Hubert H. Shook, M. D.	Ohio National, Cincinnati, Ohio
Ralph R. Simmons, M. D.	Equitable, Des Moines, Iowa
Jonathan C. Sinclair, M. D.	Canada Life, Toronto, Canada

F. Hartley Smith, M. D.	Great-West, Winnipeg, Canada
Stewart A. Smith, M. D.	Australian Mutual, Sydney, Australia
Wilbur A. Smith, M. D.	Equitable Life Assurance, New York City
Isaac Sosznitz, M. D.	Eastern, New York City
Marion Souchon, M. D.	Pan-American, New Orleans, La.
Charles G. Spivey, M. D.	Carolina Life, Columbia, S. C.
Frank L. Springer, M. D.	Columbian National, Boston, Mass.
H. Frank Starr, M. D.	Jefferson Standard, Greensboro, N. C.
F. R. Stearns, M. D.	Security Benefit, Topeka, Kan.
George G. Stebbins, M. D.	Wisconsin Life, Madison, Wis.
John B. Steele, M. D.	Volunteer State, Chattanooga, Tenn.
David F. R. Steuart, M. D.	Mutual Benefit, Newark, N. J.
Edgar M. Stevenson, M. D.	State Farm, Bloomington, Ill.
Hector M. Stevenson, M. D.	Aetna, Hartford, Conn.
Lester Q. Stewart, M. D.	Aetna, Hartford, Conn.
I. Read Stidger, M. D.	Prudential, Newark, N. J.
Frank M. Stites, M. D.	Kentucky Home Mutual, Louisville, Ky.
Nicholas A. Sullo, M. D.	Equitable Life Assurance, New York City.
Bion C. Syverson, M. D.	Equitable Life Assurance, New York City
John C. Talbot, M. D.	Pacific Mutual, Los Angeles, Calif.
Joseph L. Tansey, M. D.	John Hancock Mutual, Boston, Mass.

## LIST OF MEMBERS

321

Louis J. Tedesco, M. D.	New York Life, New York City
Gamber F. Tegtmeyer, M. D.	Northwestern Mutual, Milwaukee, Wis.
Edward R. Thompson, M. D.	Texas Prudential, Galveston, Tex.
K. Jefferson Thomson, M. D.	Metropolitan, New York City
William B. Thornton, M. D.	Norwich Union, Toronto, Canada
Joel E. Toothaker, M. D.	Sunset Life, Olympia, Wash.
Albert R. Tormey, M. D.	National Guardian, Madison, Wis.
Grafton D. Townshend, M. D.	Standard Life Association, Lawrence, Kansas
John M. Trapnell, Jr., M. D.	Penn Mutual, Philadelphia, Pa.
Joseph Travenick, Jr., M. D.	Occidental, Los Angeles, Calif.
Sophie C. Trent, M. D.	Connecticut General, Hartford, Conn.
Wallace Troup, M. D.	Metropolitan, New York City
Francis D. Truax, M. D.	Crown, Toronto, Canada
Maurice Turcotte, M. D.	Industrial, Quebec, Canada
Harry E. Ungerleider, M. D.	Equitable Life Assurance, New York City
Bruce W. Vale, M. D.	Excelsior, Toronto, Canada
Euen Van Kleeck, M. D.	Travelers, Hartford, Conn.
Alexander E. Venables, M. D.	Minnesota Mutual, St. Paul, Minn.
Frederick H. Vinup, M. D.	Monumental, Baltimore, Md.
Reynold C. Voss, M. D.	Pan-American, New Orleans, La.

Proctor C. Waldo, M. D.	Washington National, Evanston, Ill.
George H. Walker, M. D.	Lincoln Liberty, Lincoln, Neb.
Dick P. Wall, M. D.	American National, Galveston, Tex.
Gordon K. Wallace, M. D.	Great American Reserve, Dallas, Tex.
Kenneth E. Ward, M. D.	Connecticut General, Hartford, Conn.
R. Vance Ward, M. D.	Montreal Life, Montreal, Canada
Frank A. Warner, M. D.	John Hancock Mutual, Boston, Mass.
Robert L. Weaver, M. D.	Penn Mutual, Philadelphia, Pa.
Jefferson Weed, M. D.	Mutual Benefit, Newark, N. J.
Stephen S. Werth, M. D.	Globe, Chicago, Ill.
Howard E. Wiley, M. D.	Southwestern, Dallas, Tex.
John A. Wilhelm, M. D.	Gulf, Jacksonville, Fla.
Alfred A. Willander, M. D.	Mutual Trust, Chicago, Ill.
Earl B. Williams, M. D.	Wisconsin National, Oshkosh, Wis.
Ennion S. Williams, M. D.	Life Insurance Co. of Virginia, Richmond, Va.
Richard L. Willis, M. D.	Mutual, New York City
Archibald C. Wilson, M. D.	Connecticut General, Hartford, Conn.
C. L. Wilson, M. D.	Empire State Mutual, Jamestown, N. Y.
Don J. Wolfram, M. D.	Jefferson National, Indianapolis, Ind.
George E. Woodford, M. D.	Home, New York City
Donald H. Woodhouse, M. D.	Sun, Montreal, Canada
Lauritz S. Ylvisaker, M. D.	Fidelity Mutual, Philadelphia, Pa.
Donald E. Yochem, M. D.	Farm Bureau, Columbus, Ohio

LIST OF MEMBERS

323

Arthur W. Young, M. D.	Sun, Montreal, Canada
George G. Young, M. D.	Central, Des Moines, Iowa
Victor H. Young, M. D.	Travelers, Hartford, Conn.
Russell W. Zinkann, M. D.	Mutual, Waterloo, Canada
Arthur R. Zintek, M. D.	Northwestern Mutual, Milwaukee, Wis.
Albert F. Zipf, M. D.	Calif.-Western States, Sacramento, Calif.

## HONORARY MEMBERS

Francis R. Dieuaide, M. D.	New York City
Arthur Hunter	New York City
Edward E. Rhodes	Newark, N. J.

## EMERITUS MEMBERS

John W. Abbott, M. D.	Worcester, Mass.
Edwin H. Allen, M. D.	Boston, Mass.
Hiram H. Amiral, M. D.	Worcester, Mass.
William B. Bartlett, M. D.	Boston, Mass.
Chester T. Brown, M. D.	Newark, N. J.
O. M. Eakins, M. D.	Pittsburgh, Pa.
Byam Hollings, M. D.	Boston, Mass.
Walter A. Jaquith, M. D.	Columbus, Ohio
George E. Kanouse, M. D.	Newark, N. J.
Francis H. McCrudden, M. D.	Boston, Mass.
William Muhlberg, M. D.	Cincinnati, Ohio
Herbert Old, M. D.	Philadelphia, Pa.
George P. Paul, M. D.	Hartford, Conn.
Charles B. Piper, M. D.	Hartford, Conn.
James M. H. Rowland, M. D.	Baltimore, Md.
Robert L. Rowley, M. D.	Hartford, Conn.
H. Crawford Scadding, M. D.	Toronto, Canada
Ernest W. Scott, M. D.	New York City
Samuel J. Streight, M. D.	Toronto, Canada
Walter E. Thornton, M. D.	Fort Wayne, Ind.
William R. Ward, M. D.	Newark, N. J.
Fred L. Wells, M. D.	Des Moines, Iowa
David E. W. Wenstrand, M. D.	Milwaukee, Wis.
Chester F. S. Whitney, M. D.	New York City
McLeod C. Wilson, M. D.	Hartford, Conn.

## COMPANIES AND THEIR REPRESENTATIVES

Acacia Mutual Life Insurance Co., Washington, D. C.	{ J. R. B. Hutchinson, M. D. J. B. Nichols, M. D.
Aetna Life Insurance Co., Hartford, Conn.	{ K. F. Brandon, M. D. J. C. Clifford, M. D. J. G. Irving, M. D. M. H. Neill, M. D. H. M. Stevenson, M. D. L. Q. Stewart, M. D.
Alliance Nationale, Montreal, Canada	Bernard Baillargeon, M. D.
All American Assurance Co., Lafayette, La.	Paul Kurzweg, Jr., M. D.
All States Life Insurance Co., Montgomery, Ala.	Bernard Mount, M. D.
American General Life Insur- ance Co., Houston, Texas	{ Ghent Graves, M. D. M. L. Graves, M. D.
American Life Insurance Co., Birmingham, Ala.	Cabot Lull, M. D.
American Mutual Life Insur- ance Co., Des Moines, Iowa.	E. B. Mountain, M. D.
American National Insurance Co., Galveston, Texas.	D. P. Wall, M. D.
American Reserve Life Insur- ance Co., Omaha, Neb.	R. A. Moser, M. D.
Atlantic Life Insurance Co., Richmond, Va.	Cullen Pitt, M. D.

Australian Mutual Provident Society, Sydney, Australia.	{ J. H. Halliday, M. D. S. A. Smith, M. D.
Baltimore Life Insurance Co., Baltimore, Md.	N. B. Cole, M. D.
Bankers Life Company, Des Moines, Iowa.	{ A. E. Johann, M. D. George McCreight, M. D. R. E. King, M. D.
Bankers Life Insurance Co. of Nebraska, Lincoln, Neb.	H. E. Flansburg, M. D.
Bankers National Life Ins. Co., Montclair, N. J.	B. T. D. Schwarz, M. D.
Berkshire Life Insurance Co., Pittsfield, Mass.	{ F. R. Congdon, M. D. Frank Harnden, M. D.
Boston Mutual Life Insurance Co., Boston, Mass.	L. B. Ellis, M. D.
Business Men's Assurance Co. of America, Kansas City, Mo.	C. B. Ahlefeld, M. D.
Calif.-Western States Life Insurance Co., Sacramento, Calif.	A. F. Zipf, M. D.
Canada Life Assurance Co., Toronto, Canada.	{ A. E. Parks, M. D. J. C. Sinclair, M. D.
Capitol Life Insurance Co. of Colorado, Denver, Colo.	J. M. Foster, M. D.
Carolina Life Insurance Co., Columbia, S. C.	C. G. Spivey, M. D.
Central Life Assurance Society, Des Moines, Iowa.	{ M. I. Olsen, M. D. G. G. Young, M. D.
Colonial Life Insurance Co., East Orange, N. J.	L. A. Pyle, M. D.

## COMPANIES AND THEIR REPRESENTATIVES 327

Columbian National Life Ins. { C. H. Kelley, M. D.  
Co., Boston, Mass. { F. L. Springer, M. D.

Columbian Mutual Life Ins. Co., Memphis, Tenn. J. P. Moss, M. D.

Columbus Mutual Life Ins. Co., Columbus, Ohio. F. M. Green, M. D.

Commonwealth Life Insurance Co., Louisville, Ky. A. S. Irving, M. D.

Companion Life Ins. Co., New York City Joseph Altman, M. D.

Confederation Life Association, { C. D. Gossage, M. D.  
Toronto, Canada. { G. W. Lougheed, M. D.

Connecticut General Life Ins. Co., Hartford, Conn. { N. J. Barker, M. D.  
L. H. Earle, Jr., M. D.  
O. G. Goldkamp, M. D.  
C. J. M. Grisdale, M. D.  
A. J. Robinson, M. D.  
S. C. Trent, M. D.  
K. E. Ward, M. D.  
A. C. Wilson, M. D.

Connecticut Mutual Life Ins. Co., Hartford, Conn. { T. M. Ebers, M. D.  
H. F. Laramore, M. D.  
R. E. Nicholson, M. D.  
H. B. Rollins, M. D.

Constitution Life Company of America, Los Angeles, Calif. Leo Gelfand, M. D.

Continental Amer. Life Ins. Co., Wilmington, Del. W. M. Gentner, M. D.

Continental Assurance Co., { H. W. Dingman, M. D.  
Chicago, Ill. { C. L. Reeder, M. D.

Continental Life Insurance Co., Toronto, Canada. S. J. N. Magwood, M. D.

Country Life Insurance Co., Chicago, Ill. J. E. Boland, M. D.

## SIXTIETH ANNUAL MEETING

Crown Life Insurance Co.,      { H. D. Delamere, M. D.  
 Toronto, Canada.                      F. D. Truax, M. D.

Dominion Life Assurance Co., { A. J. McGanity, M. D.  
 Waterloo, Canada                      H. M. Pequegnat, M. D.

Eastern Life Insurance Co.,      Isaac Sossnitz, M. D.  
 New York City

Empire Life Insurance Co.,      J. S. Delahaye, M. D.  
 Kingston, Canada

Empire State Mutual Life In-  
 surance Co., Jamestown,  
 N. Y.                                      C. L. Wilson, M. D.

Equitable Life Assurance      { E. W. Beckwith, M. D.  
 Society, New York City              Leslie Brown, M. D.  
     R. B. Cleveland, M. D.  
     George Goodkin, M. D.  
     R. S. Gubner, M. D.  
     W. C. Lamb, M. D.  
     W. J. McNamara, M. D.  
     W. M. Reynolds, M. D.  
     W. A. Smith, M. D.  
     N. A. Sullo, M. D.  
     B. C. Syverson, M. D.  
     H. E. Ungerleider, M. D.

Equitable Life Insurance Co.,      F. M. McChesney, M. D.  
 Washington, D. C.

Equitable Life Ins. Co. of      W. L. Hilliard, M. D.  
 Canada, Waterloo,  
 Canada

Equitable Life Insurance Co.      { W. O. Purdy, M. D.  
 of Iowa, Des Moines,  
 Iowa                                      R. R. Simmons, M. D.

Excelsior Life Insurance Co.,      B. W. Vale, M. D.  
 Toronto, Canada.

Farm Bureau Life Ins. Co.,      D. E. Yochem, M. D.  
 Columbus, Ohio.

## COMPANIES AND THEIR REPRESENTATIVES 329

Farmers & Bankers Life Insurance Co., Wichita, Kan.	J. L. Evans, M. D.			
Fidelity Life Assurance Co., Regina, Canada	F. D. Munroe, M. D.			
Fidelity Mutual Life Ins. Co., Philadelphia, Pa.	<table><tr><td>J. M. Peck, M. D.</td></tr><tr><td>J. T. Sheridan, M. D.</td></tr><tr><td>L. S. Ylvisaker, M. D.</td></tr></table>	J. M. Peck, M. D.	J. T. Sheridan, M. D.	L. S. Ylvisaker, M. D.
J. M. Peck, M. D.				
J. T. Sheridan, M. D.				
L. S. Ylvisaker, M. D.				
Fidelity Union Life Insurance Co., Dallas, Texas	J. T. Downs, Jr., M. D.			
Franklin Life Ins. Co., Springfield, Ill.	<table><tr><td>E. G. Baxter, M. D.</td></tr><tr><td>Frederick Fink, M. D.</td></tr></table>	E. G. Baxter, M. D.	Frederick Fink, M. D.	
E. G. Baxter, M. D.				
Frederick Fink, M. D.				
General American Life Ins. Co., St. Louis, Mo.	J. H. Ready, M. D.			
Globe Life Insurance Co., Chicago, Ill.	S. S. Werth, M. D.			
Great American Reserve In- surance Co., Dallas, Tex.	G. K. Wallace, M. D.			
Great National Life Insurance Co., Dallas, Texas	P. M. Rattan, M. D.			
Great Southern Life Ins. Co., Houston, Texas	N. P. Doak, M. D.			
Great-West Life Assur. Co., Winnipeg, Canada.	<table><tr><td>F. A. L. Mathewson, M. D.</td></tr><tr><td>W. F. H. O'Neill, M. D.</td></tr><tr><td>F. H. Smith, M. D.</td></tr></table>	F. A. L. Mathewson, M. D.	W. F. H. O'Neill, M. D.	F. H. Smith, M. D.
F. A. L. Mathewson, M. D.				
W. F. H. O'Neill, M. D.				
F. H. Smith, M. D.				
Guarantee Mutual Life Insur- ance Co., Omaha, Neb.	J. P. Donelan, M. D.			
Guaranty Income Life Insur- ance Co., Baton Rouge, La.	H. G. Riche, M. D.			
Guardian Life Insurance Co. of America, New York City	<table><tr><td>M. B. Bender, M. D.</td></tr><tr><td>Phillips Lambkin, M. D.</td></tr><tr><td>D. C. Roberts, M. D.</td></tr></table>	M. B. Bender, M. D.	Phillips Lambkin, M. D.	D. C. Roberts, M. D.
M. B. Bender, M. D.				
Phillips Lambkin, M. D.				
D. C. Roberts, M. D.				

Gulf Life Insurance Co., Jacksonville, Fla.	J. A. Wilhelm, M. D.			
Hawaiian Life Insurance Co., Ltd., Honolulu, T. H.	C. E. Fronk, M. D.			
Home Friendly Insurance Co., Baltimore, Md.	M. Theodore Boss, M. D.			
Home Life Insurance Co., New York City	<table><tr><td>J. H. Humphries, M. D.</td></tr><tr><td>V. L. Karren, M. D.</td></tr><tr><td>G. E. Woodford, M. D.</td></tr></table>	J. H. Humphries, M. D.	V. L. Karren, M. D.	G. E. Woodford, M. D.
J. H. Humphries, M. D.				
V. L. Karren, M. D.				
G. E. Woodford, M. D.				
Home Life Ins. Co. of America, Philadelphia, Pa.	H. W. Goos, M. D.			
Home State Life Insurance Co., Oklahoma City, Okla.	W. W. Rucks, M. D.			
Illinois Bankers Life Assur- ance Co., Monmouth, Ill.	H. G. Ebersole, M. D.			
Imperial Life Assurance Co., Toronto, Canada.	<table><tr><td>R. W. Mann, M. D.</td></tr><tr><td>D. L. Selby, M. D.</td></tr></table>	R. W. Mann, M. D.	D. L. Selby, M. D.	
R. W. Mann, M. D.				
D. L. Selby, M. D.				
Independent Order of Forest- ers, Toronto, Canada	C. B. Parker, M. D.			
Indianapolis Life Ins. Co., Indianapolis, Ind.	R. M. Nay, M. D.			
Industrial Life Insurance Co., Quebec, Canada	Maurice Turcotte, M. D.			
Interstate Life and Accident Co., Chattanooga, Tenn.	J. W. Johnson, Jr., M. D.			
Jefferson National Life Insurance Co., Indianapolis, Ind.	D. J. Wolfram, M. D.			
Jefferson Standard Life Ins. Co., Greensboro, N. C.	H. F. Starr, M. D.			

COMPANIES AND THEIR REPRESENTATIVES 331

John Hancock Mutual Life Ins. Co., Boston, Mass.	$\left\{ \begin{array}{l} \text{R. A. Behrman, M. D.} \\ \text{R. L. Candage, M. D.} \\ \text{B. L. Huntington, M. D.} \\ \text{J. S. Pearson, M. D.} \\ \text{J. L. Tansey, M. D.} \\ \text{F. A. Warner, M. D.} \end{array} \right.$
Kansas City Life Ins. Co., Kansas City, Mo.	$\left\{ \begin{array}{l} \text{G. P. Barnett, M. D.} \\ \text{J. E. Bee, M. D.} \end{array} \right.$
Kentucky Home Mutual Life Insurance Co., Louisville, Ky.	F. M. Stites, M. D.
Knights of Columbus, New Haven, Conn.	G. J. Lunz, M. D.
"La Latino-Americana", Mexico, D. F.	Ignacio Mesa, M. D.
La Nacional, Compania de Seguros Sobre la Vida, S. A., Mexico, D. F.	Aniceto Del Rio, M. D.
Liberty Life Insurance Co., Greenville, S. C.	W. S. Fewell, M. D.
Liberty National Life Ins. Co., Birmingham, Ala.	J. A. Livingston, M. D.
Life & Casualty Ins. Co. of Tennessee, Nashville, Tenn.	C. T. Kirchmaier, M. D.
Life Insurance Co. of Georgia, Atlanta, Ga.	O. E. Hanes, M. D.
Life Insurance Co. of Virginia, Richmond, Va.	$\left\{ \begin{array}{l} \text{G. M. Harwood, M. D.} \\ \text{H. M. McCue, Jr., M. D.} \\ \text{E. S. Williams, M. D.} \end{array} \right.$
Lincoln Liberty Life Ins. Co., Lincoln, Neb.	G. H. Walker, M. D.
Lincoln National Life Ins. Co., Fort Wayne, Ind.	$\left\{ \begin{array}{l} \text{G. M. Graham, M. D.} \\ \text{W. H. Scoins, M. D.} \end{array} \right.$

## SIXTIETH ANNUAL MEETING

London Life Insurance Co.,      J. T. Bowman, M. D.  
 London, Canada.      G. R. Collyer, M. D.  
                                     A. S. Graham, M. D.

Loyal Protective Life Insurance Co., Boston, Mass.      H. W. Hudson, M. D.

Maccabees (The),  
 Detroit, Mich.      H. R. John, M. D.

Manhattan Life Insurance Co.,      G. H. Barber, M. D.  
 New York City      L. G. LaPointe, M. D.

Manufacturers Life Ins. Co.,  
 Toronto, Canada.      D. J. Breithaupt, M. D.  
                             T. C. Dunlop, M. D.  
                             H. M. Gray, M. D.  
                             R. C. Montgomery, M. D.

Maritime Life Insurance Co.,  
 Halifax, Canada      G. H. Murphy, M. D.

Massachusetts Mutual Life Insurance Co.,  
 Springfield, Mass.      H. B. Brown, M. D.  
                             J. G. Forgerson, M. D.  
                             J. R. E. Morden, M. D.  
                             Gordon Ross, M. D.  
                             T. S. Sexton, M. D.

Metropolitan Life Insurance Co., New York City      R. A. Benson, M. D.  
                             C. C. Berwick, M. D.  
                             N. R. Blatherwick, M. D.  
                             E. C. Bonnett, M. D.  
                             A. W. Bromer, M. D.  
                             E. T. Dewey, M. D.  
                             R. K. Farnham, M. D.  
                             H. H. Fellows, M. D.  
                             R. W. Finegan, M. D.  
                             J. T. Geiger, M. D.  
                             J. C. Horan, M. D.  
                             A. O. Jimenis, M. D.  
                             H. B. Kidd, M. D.  
                             W. J. McConnell, M. D.  
                             G. S. Pesquera, M. D.  
                             G. P. Robb, M. D.  
                             K. J. Thomson, M. D.  
                             Wallace Troup, M. D.

## COMPANIES AND THEIR REPRESENTATIVES 333

Michigan Life Insurance Co., Detroit, Mich.	R. J. Scott, M. D.
Midland Mutual Life Insurance Co., Columbus, Ohio	P. H. Charlton, M. D.
Midland National Life Insurance Co., Watertown, S. D.	O. S. Randall, M. D.
Midwest Life Insurance Co., Lincoln, Neb.	E. W. Rowe, M. D.
Minnesota Mutual Life Insurance Co., St. Paul, Minn.	A. E. Venables, M. D.
Missouri Insurance Co., St. Louis, Mo.	C. D. Magee, M. D.
Modern Woodmen of America, Rock Island, Ill.	E. A. Anderson, M. D.
Monarch Life Assur. Co., Winnipeg, Canada	A. B. Houston, M. D.
Monarch Life Insurance Co., Springfield, Mass.	J. M. Gilchrist, M. D. L. E. Hathaway, Jr., M. D.
Montreal Life Insurance Co., Montreal, Canada	R. V. Ward, M. D.
Monumental Life Insurance Co., Baltimore, Md.	F. H. Vinup, M. D.
Mutual Benefit Life Insurance Co., Newark, N. J.	J. R. Beard, M. D. E. C. Hillman, Jr., M. D. W. A. Reiter, M. D. M. T. Ryman, M. D. D. F. Steuart, M. D. Jefferson Weed, M. D.
Mutual Life Assur. Co. of Canada, Waterloo, Canada	J. M. Livingston, M. D. J. G. Ross, M. D. R. W. Zinkann, M. D.

Mutual Life Ins. Co. of New York, New York City	$\left\{ \begin{array}{l} \text{W. Pepper Constable, M. D.} \\ \text{J. R. Gudger, M. D.} \\ \text{J. F. Moore, M. D.} \\ \text{S. A. Narins, M. D.} \\ \text{E. J. Quinn, M. D.} \\ \text{R. L. Willis, M. D.} \end{array} \right.$
Mutual Trust Life Insurance Co., Chicago, Ill.	A. A. Willander, M. D.
National Equity Life Insurance Co., Little Rock, Ark.	Alfred Kahn, Jr., M. D.
National Fidelity Life Insurance Co., Kansas City, Mo.	J. V. Bell, M. D.
National Guardian Life Insurance Co., Madison, Wis.	A. R. Tormey, M. D.
National Life & Accident Ins. Co., Nashville, Tenn.	$\left\{ \begin{array}{l} \text{B. F. Byrd, M. D.} \\ \text{G. E. Fort, M. D.} \\ \text{L. C. Miller, M. D.} \end{array} \right.$
National Life Assurance Co. of Canada, Toronto, Canada	E. C. Noble, M. D.
National Life Co., Des Moines, Iowa	L. K. Meredith, M. D.
National Life Insurance Co., Montpelier, Vt.	$\left\{ \begin{array}{l} \text{G. E. Allen, M. D.} \\ \text{H. L. Colombo, M. D.} \\ \text{A. J. Oberlander, M. D.} \end{array} \right.$
National Old Line Insurance Co., Little Rock, Ark.	R. E. McLochlin, M. D.
New England Mutual Life Ins. Co., Boston, Mass.	$\left\{ \begin{array}{l} \text{F. R. Brown, M. D.} \\ \text{M. H. Clifford, M. D.} \\ \text{H. M. Frost, M. D.} \\ \text{O. C. Hendrix, M. D.} \end{array} \right.$

COMPANIES AND THEIR REPRESENTATIVES 335

New York Life Insurance Co., New York City	D. R. Auten, M. D.
	M. F. Bell, M. D.
	William Bolt, M. D.
	E. J. Campbell, M. D.
	G. D. Dorman, M. D.
	A. H. Faber, M. D.
	E. M. Freeland, M. D.
	E. E. Getman, M. D.
	H. L. Hauge, M. D.
	T. B. Hoxie, M. D.
	J. J. Hutchinson, M. D.
	I. C. Lawler, M. D.
	John Malgieri, M. D.
	R. W. Pratt, M. D.
	L. J. Tedesco, M. D.
North American Life Assur. Co., Toronto, Canada	J. G. Falconer, M. D.
	Eugene Montgomery, M. D.
North American Reassurance Co., New York City	E. V. Higgins, M. D.
	J. T. Montgomery, M. D.
Northern Life Assurance Co. of Canada, London, Canada	J. H. Geddes, M. D.
Northwestern Mutual Life Ins. Co., Milwaukee, Wis.	R. W. Benton, M. D.
	J. M. Bond, M. D.
	J. A. End, M. D.
	R. T. Gilchrist, M. D.
	D. F. Rikkers, M. D.
	G. F. Tegtmeier, M. D.
	A. R. Zintek, M. D.
Northwestern National Life Ins. Co., Minneapolis, Minn.	K. W. Anderson, M. D.
Norwich Union Life Insurance Society, Toronto, Canada	
	W. B. Thornton, M. D.
Occidental Life Ins. Co. of California, Los Angeles, Calif.	E. F. Sheldon, M. D.
	Joseph Travenick, Jr., M. D.

Ohio National Life Ins. Co., Cincinnati, Ohio	H. H. Shook, M. D.							
Ohio State Life Insurance Co., Columbus, Ohio	T. F. Ross, M. D.							
Old Line Life Insurance Co. of America, Milwaukee, Wis.	H. M. Hawkins, M. D.							
Oriental Government Security Life Assurance Co., Ltd., Bombay, India.	K. J. J. Cursetji, M. D.							
Pacific Mutual Life Ins. Co., Los Angeles, Calif.	<table><tr><td>F. R. Anderson, M. D.</td></tr><tr><td>L. H. Lee, M. D.</td></tr><tr><td>J. C. Talbot, M. D.</td></tr></table>	F. R. Anderson, M. D.	L. H. Lee, M. D.	J. C. Talbot, M. D.				
F. R. Anderson, M. D.								
L. H. Lee, M. D.								
J. C. Talbot, M. D.								
Pan-American Life Ins. Co., New Orleans, La.	<table><tr><td>Marion Souchon, M. D.</td></tr><tr><td>R. C. Voss, M. D.</td></tr></table>	Marion Souchon, M. D.	R. C. Voss, M. D.					
Marion Souchon, M. D.								
R. C. Voss, M. D.								
Paul Revere Life Ins. Co., Worcester, Mass.	<table><tr><td>Earl J. Kuenster, M. D.</td></tr><tr><td>H. R. Leffingwell, M. D.</td></tr></table>	Earl J. Kuenster, M. D.	H. R. Leffingwell, M. D.					
Earl J. Kuenster, M. D.								
H. R. Leffingwell, M. D.								
Peninsular Life Insurance Co., Jacksonville, Fla.	E. B. Milam, M. D.							
Penn Mutual Life Ins. Co., Philadelphia, Pa.	<table><tr><td>B. A. Dawber, M. D.</td></tr><tr><td>E. S. Dillon, M. D.</td></tr><tr><td>D. W. Hoare, M. D.</td></tr><tr><td>W. R. Leute, Jr., M. D.</td></tr><tr><td>C. F. Nichols, M. D.</td></tr><tr><td>J. M. Trapnell, Jr., M. D.</td></tr><tr><td>R. L. Weaver, M. D.</td></tr></table>	B. A. Dawber, M. D.	E. S. Dillon, M. D.	D. W. Hoare, M. D.	W. R. Leute, Jr., M. D.	C. F. Nichols, M. D.	J. M. Trapnell, Jr., M. D.	R. L. Weaver, M. D.
B. A. Dawber, M. D.								
E. S. Dillon, M. D.								
D. W. Hoare, M. D.								
W. R. Leute, Jr., M. D.								
C. F. Nichols, M. D.								
J. M. Trapnell, Jr., M. D.								
R. L. Weaver, M. D.								
Pennsylvania Life, Health & Accident Ins. Co., Philadelphia, Pa.	John P. Chapman, M. D.							
Peoples Life Ins. Co., Frankfort, Ind.	C. A. Robison, M. D.							
Peoples Life Insurance Co., Washington, D. C.	<table><tr><td>J. R. Biggs, M. D.</td></tr><tr><td>M. A. Puzak, M. D.</td></tr></table>	J. R. Biggs, M. D.	M. A. Puzak, M. D.					
J. R. Biggs, M. D.								
M. A. Puzak, M. D.								
Philadelphia Life Ins. Co., Philadelphia, Pa.	T. M. Armstrong, M. D.							

## COMPANIES AND THEIR REPRESENTATIVES 337

Phoenix Mutual Life Ins. Co., Hartford, Conn.	{ H. B. Campbell, M. D. R. A. Goodell, M. D. Llewellyn Hall, M. D. N. R. Kelley, M. D.
Pilot Life Insurance Co., Greensboro, N. C.	J. L. Cook, M. D.
Pioneer Mutual Life Insur- ance Co., Fargo, N. D.	F. O. Gronvold, M. D.
Policyholder's National Life Ins. Co., Sioux Falls, S. D.	Rezin Reagan, M. D.
Postal Life Insurance Co., New York City	L. B. Dunn, M. D.
Praetorians (The), Dallas, Texas	E. P. Leeper, M. D.
Preferred Life Assurance So- ciety, Montgomery, Ala.	B. C. Bird, M. D.
Protected Home Circle, Sharon, Pa.	W. G. McLaughry, M. D.
Protective Life Insurance Co., Birmingham, Ala.	E. G. Givhan, Jr., M. D.
Provident Life and Accident Ins. Co., Chattanooga, Tenn.	W. R. Bishop, M. D.
Provident Mutual Life Ins. Co., Philadelphia, Pa.	{ E. J. Dewees, M. D. P. H. Langner, Jr., M. D. L. L. McLellan, M. D. D. S. Pepper, M. D.
Prudential Assur. Co., Ltd., Montreal, Canada	{ E. S. Mills, M. D. C. A. Peters, M. D.

## SIXTIETH ANNUAL MEETING

Prudential Insurance Co. of America, Newark, N. J.

R. F. Buchan, M. D.  
 P. E. Carlisle, M. D.  
 E. G. Dewis, M. D.  
 A. H. Domm, M. D.  
 R. L. Dross, M. D.  
 F. I. Ganot, M. D.  
 A. E. Gras, M. D.  
 W. C. Hausheer, M. D.  
 E. G. Howe, M. D.  
 E. A. Keenleyside, M. D.  
 C. E. Kiessling, M. D.  
 H. B. Kirkland, M. D.  
 J. C. Lindner, M. D.  
 F. J. McGurl, M. D.  
 R. A. Nelson, M. D.  
 W. C. Page, M. D.  
 P. V. Reinartz, M. D.  
 K. F. Schaefer, M. D.  
 I. R. Stidger, M. D.

Puritan Life Insurance Co., Providence, R. I.

E. D. Chesebro, M. D.

Pyramid Life Insurance Co., Little Rock, Ark.

J. H. Sanderlin, M. D.

Reliance Insurance Co. of Pittsburgh, Pittsburgh, Pa.

R. L. Anderson, Jr., M. D.  
 H. A. Cochran, Jr., M. D.  
 I. Kenneth Gardner, M. D.  
 J. L. Humphreys, M. D.

Republic National Life Ins. Co., Dallas, Texas

J. E. Hunsinger, M. D.  
 D. G. Kilgore, M. D.

Rockford Life Insurance Co., Rockford, Ill.

P. A. Anderson, M. D.

Royal Insurance Co., Ltd., Montreal, Canada

G. W. Halpenny, M. D.

Security Benefit Life Ins. Co., Topeka, Kan.

F. R. Stearns, M. D.

Security Life and Accident Co., Denver, Colo.

D. S. Baughman, M. D.  
 R. C. Scannell, M. D.

## COMPANIES AND THEIR REPRESENTATIVES 339

Security Life & Trust Co., Winston-Salem, N. C.	S. W. Hurdle, M. D.
Security Mutual Life Ins. Co., Binghamton, N. Y.	W. B. Aten, M. D. V. G. Hammond, M. D.
Shenandoah Life Insurance Co., Inc., Roanoke, Va.	D. S. Garner, M. D.
Southern Life Insurance Co. of Georgia, Atlanta, Ga.	D. Y. Sage, M. D.
Southland Life Insurance Co., Dallas, Texas	Hall Shannon, M. D.
Southwestern Life Ins. Co., Dallas, Texas	C. F. Brown, M. D. H. E. Wiley, M. D.
Sovereign Life Assurance Co., Winnipeg, Canada	C. P. Neilson, M. D.
Standard Insurance Company, Portland, Oregon	E. L. Boylen, M. D.
Standard Life Association Lawrence, Kansas	G. D. Townshend, M. D.
Standard Life Assur. Co., Montreal, Canada	W. W. Eakin, M. D.
State Farm Life Insurance Co., Bloomington, Ill.	J. K. P. Hawks, M. D. E. M. Stevenson, M. D.
State Life Insurance Co., Indianapolis, Ind.	F. T. Hallam, M. D.
State Mutual Life Assur. Co., Worcester, Mass.	F. P. Bicknell, M. D. C. C. Beach, M. D. E. A. Reiman, M. D.
State Reserve Life Insurance Co., Fort Worth, Texas	Samuel Jagoda, M. D.

Sun Life Assurance Company of Canada, Montreal, Canada	J. K. Gordon, M. D. D. H. Woodhouse, M. D. A. W. Young, M. D.
Sun Life Insurance Co. of America, Baltimore, Md.	George McLean, M. D.
Sunset Life Insurance Co. of America, Olympia, Wash.	J. E. Toothaker, M. D.
Teachers Insurance & Annuity Association, New York City	William MacDonald, M. D.
T. Eaton Life Assurance Co., Toronto, Canada	C. V. Mulligan, M. D.
Texas Life Insurance Co.. Waco, Texas	I. E. Colgin, M. D.
Texas Prudential Insurance Co., Galveston, Texas	E. R. Thompson, M. D.
Toronto Mutual Life Ins. Co., Toronto, Canada	J. A. A. Harcourt, M. D.
Travelers Insurance Company, Hartford, Conn.	J. T. Cabaniss, M. D. R. M. Filson, M. D. A. L. Larson, M. D. A. F. Seibert, M. D. Euen van Kleeck, M. D. V. H. Young, M. D.
Union Central Life Insurance Co., Cincinnati, Ohio	W. D. Hickerson, M. D. Edward Kuck, M. D. Charles Maertz, M. D.
Union Labor Life Insurance Co., New York City	W. L. O'Connell, M. D.
Union Life Insurance Co., Little Rock, Ark.	J. H. Hayes, M. D.
Union Mutual Life Insurance Co., Portland, Maine	H. E. Christensen, M. D.
United Benefit Life Insurance Co., Omaha, Neb.	N. L. Criss, M. D.

## COMPANIES AND THEIR REPRESENTATIVES 341

United Fidelity Life Insurance Co., Dallas, Texas	H. K. Crutcher, M. D.
United Life and Accident Ins. Co., Concord, N. H.	H. H. Amsden, M. D. P. M. L. Forsberg, M. D.
United States Life Ins. Co., New York City	J. A. Avrack, M. D.
Volunteer State Life Ins. Co., Chattanooga, Tenn.	J. B. Steele, M. D.
Washington National Insur- ance Company, Evanston, Ill.	I. E. Hoffman, M. D. P. C. Waldo, M. D.
West Coast Life Ins. Co., San Francisco, Calif.	I. C. Heron, M. D.
Western Life Assurance Company, Hamilton, Canada	G. E. Greenway, M. D.
Western Life Insurance Company, Helena, Mont.	T. L. Hawkins, M. D. E. H. Lindstrom, M. D.
Western Reserve Life Insurance Company, Austin, Texas	C. P. Hardwicke, M. D.
Western and Southern Life Ins. Co., Cincinnati, Ohio	C. M. Barrett, M. D. E. R. Bush, M. D.
Western States Life Insurance Company, Fargo, N. D.	T. H. Lewis, M. D.
Wisconsin Life Insurance Company, Madison, Wis.	G. G. Stebbins, M. D.
Wisconsin National Life Insurance Company, Oshkosh, Wis.	E. B. Williams, M. D.
Woodmen of the World Life Insurance Society, Omaha, Neb.	H. B. Kennedy, M. D.

## AUTHOR INDEX

Vol. XXXV—1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Page
Andrews, James, Jr.: Relationships Between the Medical Profession and the Health Insurance Council .....	162
Bell, Murray F.: Calcification of the Thoracic Aorta: A Mortality Study .....	135
Bolt, William: Calcification of the Thoracic Aorta: A Mortality Study .....	135
Bonnett, Earl C.: <i>Mortality Among Insured Overweights in Recent Years</i> .....	263
Buchan, Ronald F.: The Impact of Life Insurance on Public Health	201
Dieuaide, Francis R.: Current Progress in Cardiovascular Research	57
Dillon, Edward S.: Overweight as a Contributing Factor in the Development of Diabetes and Its Complications .....	280
Dublin, Louis I.: Mortality Among Insured Overweights in Recent Years .....	235
Durant, Thomas M.: Insurance Hazards of Overweight: Dietary Factors in the Development of Atherosclerosis .....	267
Falconer, J. Gilbert: Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis .....	43
Filson, Ralph: <i>Relationships Between the Medical Profession and the Health Insurance Council</i> .....	169
Gudger, James R.: The Life Insurance Examiner and the Cardiovascular System .....	149
Hench, Philip S.: Cortisone, Hydrocortisone and Corticotropin: Some Facts and Speculations with Special Reference to Rheumatoid Arthritis .....	5
Hilleboe, Herman E.: The Public Health Situation Today: Public Health and Civil Defense .....	171
Ingelfinger, Franz J.: The Prognosis of Benign Gastrointestinal Conditions .....	206
Kiessling, Charles E.: The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks .....	86
<i>Calcification of the Thoracic Aorta: A Mortality Study</i> .....	144

## AUTHOR INDEX — Continued

Vol. XXXV—1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Page
Kirkland, Henry B.: The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks ..	86
Langner, Paul H.: <i>The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks</i> ..	133
Lee, Linford H.: Remarks .....	299
Lyle, Annie Mary: The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks ..	86
Marks, Herbert H.: Mortality Among Insured Overweights in Recent Years .....	235
<i>Overweight as a Contributing Factor in the Development of Hypertension</i> .....	296
Marvin, H. M.: The Differential Diagnosis of Chest Pain .....	64
McCue, Howard M., Jr.: The Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease .....	35
McLellan, Lawrence L.: Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers .....	224
Peck, John McC.: Overweight as a Contributing Factor in the Development of Hypertension .....	291
Pepper, D. Sergeant: Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers .....	224
<i>The Prognosis of Benign Gastrointestinal Conditions</i> .....	231
Sheridan, Joyce T.: Overweight as a Contributing Factor in the Development of Hypertension .....	291
Sinclair, Jonathan C.: <i>Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis</i> .....	53
Trapnell, John M., Jr.: Overweight as a Contributing Factor in the Development of Diabetes and Its Complications .....	280
Ungerleider, Harry E.: <i>Relationships Between the Medical Profession and the Health Insurance Council</i> .....	169
Wheatley, George M.: Some Contributions of Public Health to Life Insurance .....	189
Williams, Ennion S.: <i>The Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease</i> .....	41
Ylvisaker, Lauritz S.: President's Address .....	1

## SUBJECT INDEX

Vol. XXXV—1951

Note—The Names of Authors are printed in Plain Type;  
of Discussers, in *Italics*

	Page
Address: Presidential. Ylvisaker .....	1
Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis. Falconer, <i>Sinclair</i> .....	43
Calcification of the Thoracic Aorta: A Mortality Study. Bolt, Bell, <i>Kiessling</i> .....	135
Cortisone, Hydrocortisone and Corticotropin: Some Facts and Speculations with Special Reference to Rheumatoid Arthritis. Hench .....	5
Current Progress in Cardiovascular Research. Dieuaide .....	57
Differential Diagnosis of Chest Pain. Marvin .....	64
Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks. Kirkland, Kiessling, Lyle, <i>Langer</i> .....	86
Impact of Life Insurance on Public Health. Buchan .....	201
Insurance Hazards of Overweight: Dietary Factors in the Development of Atherosclerosis. Durant .....	267
Life Insurance Examiner and the Cardiovascular System. Gudger ..	149
Members of Association:	
Active, 305; Companies and Representatives, 325; Deceased since Fifty-Ninth Annual Meeting, 304; Emeritus, 324; Honorary, 324	
Mortality Among Insured Overweights in Recent Years. Dublin, Marks, <i>Bonnett</i> .....	235
Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease. McCue, <i>Williams</i> .....	35
Overweight as a Contributing Factor in the Development of Diabetes and Its Complications. Dillon, Trapnell .....	280
Overweight as a Contributing Factor in the Development of Hypertension. Sheridan, Peck, <i>Marks</i> .....	291
Prognosis of Benign Gastrointestinal Conditions. Ingelfinger, <i>Pepper</i> 206	
Public Health Situation Today: Public Health and Civil Defense. Hilleboe .....	171
Relationships Between the Medical Profession and the Health Insurance Council. Andrews, <i>Filson, Ungerleider</i> .....	162
Remarks. Lee .....	299
Some Contributions of Public Health to Life Insurance. Wheatley ..	189
Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers. McLellan, <i>Pepper</i> 224	

## CUMULATIVE AUTHOR INDEX

1947—1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Volume	Page
Andrews, James, Jr.: Relationships Between the Medical Profession and the Health Insurance Council .....	XXXV	162
Bell, Murray F.: Calcification of the Thoracic Aorta: A Mortality Study .....	XXXV	135
Prognostic Import of a Large $Q_3$ Deflection—A Mortality Study .....	XXXIV	87
Birchard, Cecil C.: The Disease Called Arterial Hypertension—The Newer Ideas .....	XXXIII	112
Blatherwick, Norman R.: Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test .....	XXXI	5
Bolt, William: Calcification of the Thoracic Aorta: A Mortality Study .....	XXXV	135
Prognostic Import of a Large $Q_3$ Deflection—A Mortality Study .....	XXXIV	87
Bonnett, Earl C.: <i>Mortality Among Insured Overweights in Recent Years</i> .....	XXXV	263
Brandon, Kenneth F.: The Electrocardiogram in Insurance Selection .....	XXXIV	143
Buchan, Ronald F.: The Impact of Life Insurance on Public Health .....	XXXV	201
Campbell, Hugh B.: Sarcoidosis and Histoplasmosis .....	XXXIV	111
Chamberlain, W. Edward: Applications of Atomic Energy to Biology and Medicine .....	XXXII	59
Coley, Bradley L.: Prognosis in Tumors of Bone and Sarcomas of Soft Tissue .....	XXXI	73
Constable, W. Pepper: Myocardial Infarction—A Mortality Study .....	XXXIV	69
Denker, Peter G.: Epilepsy (Open Forum) .....	XXXII	116
Dewis, Edwin G.: President's Address .....	XXXIII	1
Dieuaide, Francis R.: Current Progress in Cardiovascular Research .....	XXXV	57
Report—Life Insurance Medical Research Fund .....	XXXIV	1

## CUMULATIVE AUTHOR INDEX — Continued

1947 — 1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Volume	Page
Dillon, Edward S.: Overweight as a Contributing Factor in the Development of Diabetes and Its Complications	XXXV	280
Dingman, Harry W.: <i>Pulmonary Tuberculosis Mortality — A Study of Disability Claims</i> .....	XXXII	109
Dock, William: Prophylaxis and Therapy of Arteriosclerosis .....	XXXIV	4
Donelan, James P.: Orthopedic Impairments (Open Forum) .....	XXXII	122
Dublin, Louis I.: Mortality Among Insured Overweights in Recent Years .....	XXXV	235
Durant, Thomas M.: Insurance Hazards of Overweight: Dietary Factors in the Development of Atherosclerosis	XXXV	267
Falconer, J. Gilbert: Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis .....	XXXV	43
Chronic Cholecystitis (Open Forum) .....	XXXII	136
Fellows, Haynes H.: Relationship of X-ray and Tuberculosis in Underwriting .....	XXXI	95
Filson, Ralph M.: <i>Claims</i> (Open Forum) .....	XXXI	146
Relationships Between the Medical Profession and the Health Insurance Council .....	XXXV	169
Finegan, Rexford W.: Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test .....	XXXI	5
Gross, Robert E.: Surgical Treatment in One Hundred and Thirty Cases of Coarctation of the Aorta .....	XXXIII	83
Gubner, Richard S.: The Diagnosis of Arteriosclerosis ..	XXXIV	20
Gudger, James R.: The Life Insurance Examiner and the Cardiovascular System .....	XXXV	149
Harrison, Tinsley R.: Degenerative Diseases: A Backward and a Forward Look .....	XXXII	12
Hawley, Paul R.: Voluntary Health Insurance, Its Successes and Its Failures to Date .....	XXXIII	35
Heller, Ralph T.: <i>Claims</i> (Open Forum) .....	XXXI	152

## CUMULATIVE AUTHOR INDEX — Continued

1947—1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Volume	Page
Helpern, Milton: Sudden and Unexpected Natural Death	XXXI	131
Hench, Philip S.: Cortisone, Hydrocortisone and Corticotropin: Some Facts and Speculations with Special Reference to Rheumatoid Arthritis	XXXV	5
Hilleboe, Herman E.: The Public Health Situation Today: Public Health and Civil Defense	XXXV	171
Ingelfinger, Franz J.: The Prognosis of Benign Gastrointestinal Conditions	XXXV	206
Jimenis, Albert O.: Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test	XXXI	5
President's Address	XXXI	1
Johnson, Joseph E.: The Present Status of Antimicrobial Therapy	XXXIII	73
Joslin, Elliott P.: Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test	XXXI	36
Kelly, John G.: Claims (Open Forum)	XXXI	159
Kiessling, Charles E.: Calcification of the Thoracic Aorta: A Mortality Study	XXXV	144
Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks	XXXV	86
Selection of Individuals with a Personal History of Tuberculosis on the Basis of a Single Chest X-ray	XXXI	91
Kirkland, Henry B.: The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks	XXXV	86
Lahey, Frank H.: Lesions of Terminal Ileum, Colon, and Rectum	XXXI	111
Langner, Paul H.: The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks	XXXV	133
Lee, Linford H.: Remarks	XXXV	299

## CUMULATIVE AUTHOR INDEX — Continued

1947 — 1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Volume	Page
Lew, Edward A.: <i>Pulmonary Tuberculosis Mortality—A Study of Disability Claims</i> .....	XXXII	103
Lyle, Annie Mary: The Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks .....	XXXV	86
Marks, Herbert H.: Mortality Among Insured Overweights in Recent Years .....	XXXV	235
Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test .....	XXXI	5
<i>Overweight as a Contributing Factor in the Development of Hypertension</i> .....	XXXV	296
Marvin, H. M.: The Differential Diagnosis of Chest Pain .....	XXXV	64
McAlister, H. Clive: <i>Ulcers of the Duodenum</i> .....	XXXII	179
Underwriting the Highly Substandard Risk .....	XXXIV	132
McCue, Howard M., Jr.: The Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease ..	XXXV	35
McDermott, Walsh: The Present Status of Antimicrobial Therapy .....	XXXIII	57
McGanity, Arthur J.: <i>Open Forum, Moderator</i> .....	XXXI	145
McLellan, Lawrence L.: Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers .....	XXXV	224
McMahon, The Honorable Brien: The Struggle for Atomic Peace .....	XXXII	76
Middleton, William S.: Rickettsial Diseases in the United States .....	XXXIII	6
Miscall, Laurence: <i>Surgical Treatment in One Hundred and Thirty Cases of Coarctation of the Aorta</i> .....	XXXIII	92
Neill, Mather H.: The Electrocardiogram in Insurance Selection .....	XXXIV	143
Page, Irvine H.: The Nature and Treatment of Hypertension .....	XXXI	48

## CUMULATIVE AUTHOR INDEX—Continued

1947—1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Volume	Page
Parker, Herbert M.: Insurability of Atomic Energy Workers .....	XXXII	42
Peck, John McC.: Overweight as a Contributing Factor in the Development of Hypertension .....	XXXV	291
Pepper, D. Sergeant: <i>The Prognosis of Benign Gastro-intestinal Conditions</i> .....	XXXV	231
Vagotomy and Subtotal Gastrectomy: Effect on Insurability of Individuals with Gastric and Duodenal Ulcers .....	XXXV	224
Pepper, O. H. Perry: Medical Follow-up Studies of Veterans .....	XXXIII	99
Rees, H. Maynard: <i>Claims</i> (Open Forum) .....	XXXI	165
Reisner, David: The Roentgenogram as an Aid in the Disposition of Cases of Pulmonary Tuberculosis Detected in Group Surveys .....	XXXI	85
Rhoads, C. P.: Survival of Patients with Cancer as a Function of Research .....	XXXIII	44
Robinson, Albert J.: President's Address .....	XXXII	1
Rusk, Howard A.: Dynamic Therapeutics in Chronic Disease Pays Dividends .....	XXXII	158
Rynearson, Edward H.: Is Obesity an Endocrine Problem? .....	XXXIV	99
Schwarz, Berthold T. D.: Prognosis in Asthma: An Etiologic Classification (Open Forum) .....	XXXII	140
Scott, Roy W.: Prognosis in Coronary Artery Disease	XXXIV	56
Shepherd, Pearce: Pulmonary Tuberculosis Mortality—A Study of Disability Claims .....	XXXII	85
Sheridan, Joyce T.: Overweight as a Contributing Factor in the Development of Hypertension .....	XXXV	291
Simmons, Ralph R.: <i>Open Forum, Moderator</i> .....	XXXII	115
Sinclair, Jonathan C.: <i>Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis</i> .....	XXXV	53
Smith, Alan DeForest: Some of the Causes of Low Back Pain .....	XXXIII	23

## CUMULATIVE AUTHOR INDEX—Continued

1947—1951

Note—Titles of Original Papers in Plain Type;  
of Papers Discussed, in *Italics*

	Volume	Page
Streeter, Gordon C.: The Electrocardiogram in Insurance Selection .....	XXXIV	143
Trapnell, John M., Jr.: Overweight as a Contributing Factor in the Development of Diabetes and Its Complications .....	XXXV	280
Ungerleider, Harry E.: President's Remarks .....	XXXIV	159
<i>Relationships Between the Medical Profession and the Health Insurance Council</i> .....	XXXV	169
Waldron, Frederick A.: Myocardial Infarction—A Mortality Study .....	XXXIV	69
Weaver, Robert L.: Osteomyelitis (Open Forum) .....	XXXII	128
Wheatley, George M.: Some Contributions of Public Health to Life Insurance .....	XXXV	189
Wilde, Frazar B.: A Layman Looks at the Medical Department .....	XXXII	4
Williams, Ennion S.: <i>The Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease</i> .....	XXXV	41
Wilson, McLeod C.: Ulcers of the Duodenum .....	XXXII	169
Ylvisaker, Lauritz S.: <i>Degenerative Diseases: A Backward and a Forward Look</i> .....	XXXII	12
President's Address .....	XXXV	1
Remarks .....	XXXIV	159

## CUMULATIVE SUBJECT INDEX

1947—1951

Note—The Names of Authors are Printed in Plain Type;  
of Discussers, in *Italics*

	Volume	Page
Address: Presidential. Dewis .....	XXXIII	1
Jimenis .....	XXXI	1
Robinson .....	XXXII	1
Ylvisaker .....	XXXV	1
Antimicrobial Therapy, Present Status of. McDermott, <i>Johnson</i> .....	XXXIII	57
Applications of Atomic Energy to Biology and Medicine. <i>Chamberlain</i> .....	XXXII	59
Arteriosclerosis, The Diagnosis of. Gubner .....	XXXIV	20
Arteriosclerosis, Prophylaxis and Therapy of. Dock .....	XXXIV	4
Asthma, Prognosis in, An Etiologic Classification (Open Forum). Schwarz .....	XXXII	140
Blood Dyscrasias: Current Concepts: The Effect of Treatment on Prognosis. Falconer, <i>Sinclair</i> .....	XXXV	43
Calcification of the Thoracic Aorta: A Mortality Study. <i>Bolt, Bell, Kiessling</i> .....	XXXV	135
Chronic Cholecystitis (Open Forum). Falconer .....	XXXII	136
Coarctation of the Aorta, Surgical Treatment in One Hundred and Thirty Cases of. Gross, <i>Miscall</i> .....	XXXIII	83
Coronary Artery Disease, Prognosis in. Scott .....	XXXIV	56
Cortisone, Hydrocortisone and Corticotropin: Some Facts and Speculations with Special Reference to Rheuma- toid Arthritis. Hench .....	XXXV	5
Current Progress in Cardiovascular Research. Dieuaide .....	XXXV	57
Degenerative Diseases: A Backward and a Forward Look. <i>Harrison, Ylvisaker</i> .....	XXXII	12
Differential Diagnosis of Chest Pain. Marvin .....	XXXV	64
Disease Called Arterial Hypertension—The Newer Ideas. Birchard .....	XXXIII	112
Dynamic Therapeutics in Chronic Disease Pays Dividends. Rusk .....	XXXII	158

## CUMULATIVE SUBJECT INDEX — Continued

1947 — 1951

Note—The Names of Authors are Printed in Plain Type;  
of Discussers, in *Italics*

	Volume	Page
Electrocardiogram in Insurance Selection. Brandon, Neill and Streeter .....	XXXIV	143
Epilepsy (Open Forum). Denker .....	XXXII	116
Evaluation of Certain Fundamental Electrocardiographic Patterns in the Selection of Insurance Risks. Kirkland, Kiessling, Lyle, <i>Langner</i> .....	XXXV	86
Hypertension, Nature and Treatment of. Page .....	XXXI	48
Impact of Life Insurance on Public Health. Buchan ....	XXXV	201
Insurability of Atomic Energy Workers. Parker .....	XXXII	42
Insurance Hazards of Overweight: Dietary Factors in the Development of Atherosclerosis. Durant .....	XXXV	267
Layman Looks at The Medical Department. Wilde ....	XXXII	4
Lesions of Terminal Ileum, Colon, and Rectum. Lahey ..	XXXI	111
Life Insurance Examiner and the Cardiovascular System. Gudger .....	XXXV	149
Low Back Pain, Some of the Causes of. Smith .....	XXXIII	23
Medical Follow-up Studies of Veterans. Pepper .....	XXXIII	99
Members of the Association: Active. XXXI, 185; XXXII, 206; XXXIII, 133; XXXIV, 164; XXXV, 305.		
Members of the Association: Companies and Representatives. XXXI, 204; XXXII, 225; XXXIII, 152; XXXIV, 183; XXXV, 325.		
Members of the Association: Deceased. XXXI, 179; XXXII, 200; XXXIII, 127.		
Members of the Association: Deceased since Last Annual Meeting. XXXI, 178; XXXII, 199; XXXIII, 126; XXXIV, 163; XXXV, 304.		
Members of the Association: Emeritus. XXXI, 203; XXXII, 224; XXXIII, 151; XXXIV, 182; XXXV, 324.		
Members of the Association: Honorary. XXXI, 203; XXXII, 224; XXXIII, 151; XXXIV, 182; XXXV, 324.		

## CUMULATIVE SUBJECT INDEX — Continued

1947 — 1951

Note—The Names of Authors are Printed in Plain Type;  
of Discussers, in *Italics*

	Volume	Page
Mortality Among Insured Overweights in Recent Years. Dublin, Marks, <i>Bonnett</i> .....	XXXV	235
Mortality Study of Applicants for Insurance Given a Glucose Tolerance Test. Jimenis, Marks, Finegan, Blatherwick, <i>Joslin</i> .....	XXXI	5
Myocardial Infarction: A Mortality Study. Waldron, Constable .....	XXXIV	69
Obesity, An Endocrine Problem? Rynearson .....	XXXIV	99
Open Forum, McGanity, Moderator Claims. <i>Filson, Heller, Kelly, Rees</i> .....	XXXI	145
Simmons, Moderator Problems of Medical Selection. Denker, Donelan, Weaver, Falconer, Schwarz .....	XXXII	115
Orthopedic Impairments (Open Forum). Donelan .....	XXXII	122
Osteomyelitis (Open Forum). Weaver .....	XXXII	128
Outlook for the Control of Rheumatic Fever and Rheumatic Heart Disease. McCue, <i>Williams</i> .....	XXXV	35
Overweight as a Contributing Factor in the Development of Diabetes and Its Complications. Dillon, Trapnell	XXXV	280
Overweight as a Contributing Factor in the Development of Hypertension. Sheridan, Peck, <i>Marks</i> .....	XXXV	291
Prognosis in Tumors of Bone and Sarcomas of Soft Tissue. Coley .....	XXXI	73
Prognosis of Benign Gastrointestinal Conditions. Ingelfinger, <i>Pepper</i> .....	XXXV	206
Public Health Situation Today: Public Health and Civil Defense. Hilleboe .....	XXXV	171
Pulmonary Tuberculosis Mortality—A Study of Dis- ability Claims. Shepherd, <i>Lew, Dingman</i> .....	XXXII	85
Q <sub>3</sub> Deflection, Large, Prognostic Import of: A Mortality Study. Bolt, Bell .....	XXXIV	87
Relationships Between the Medical Profession and the Health Insurance Council. Andrews, <i>Filson, Un- gerleider</i> .....	XXXV	162

## CUMULATIVE SUBJECT INDEX — Continued

1947 — 1951

Note—The Names of Authors are Printed in Plain Type;  
of Discussers, in *Italics*

	Volume	Page
Remarks: Lee .....	XXXV	299
Ungerleider .....	XXXIV	159
Ylvisaker .....	XXXIV	159
Report on Life Insurance Medical Research Fund.		
Dieuaide .....	XXXIV	1
(see Current Progress in Cardiovascular Research.		
Dieuaide) .....	XXXV	57
Rickettsial Diseases in the United States. Middleton .....	XXXIII	6
Roentgenogram as an Aid in the Disposition of Cases of		
Pulmonary Tuberculosis Detected in Group Surveys.		
Reisner .....	XXXI	85
Sarcoidosis and Histoplasmosis. Campbell .....	XXXIV	111
Selection of Individuals with a Personal History of		
Tuberculosis on the Basis of a Single Chest X-ray.		
Kiessling .....	XXXI	91
Some Contributions of Public Health to Life Insurance.		
Wheatley .....	XXXV	189
Struggle for Atomic Peace. McMahon .....	XXXII	76
Sudden and Unexpected Natural Death. Helpern .....	XXXI	131
Survival of Patients with Cancer as a Function of		
Research. Rhoads .....	XXXIII	44
Ulcers of the Duodenum. Wilson, <i>McAlister</i> .....	XXXII	169
Underwriting the Highly Substandard Risk. <i>McAlister</i> ..	XXXIV	132
Vagotomy and Subtotal Gastrectomy: Effect on In-		
surability of Individuals with Gastric and Duodenal		
Ulcers. McLellan, Pepper .....	XXXV	224
Voluntary Health Insurance, Its Successes and Its		
Failures to Date. Hawley .....	XXXIII	35
X-ray and Tuberculosis in Underwriting, Relationship of.		
Fellows .....	XXXI	95

